88-00376/

The Industrial Disease Standards Panel is a Schedule 1 Agency of the Government of Ontario attached to the Ministry of Labour. The function of the Panel, as defined in Section 86p of the Workers' Compensation Act of Ontario, is as follows:

(a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases: and

(d) to advise on eligibility rules regarding compensation for claims respecting industrial diseases.

The Panel is required by statute to report its findings to the Workers' Compensation Board of Ontario.

Additional copies of this publication are available by writing:

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ISBN 0-7729-2901-7

REPORT TO THE WORKERS' COMPENSATION BOARD
ON
THE ONTARIO GOLD MINING INDUSTRY

Industrial Disease Standards Panel

IDSP Report No. 1

Toronto, Ontario

April, 1987

REPORT TO THE WORKERS' COMPENSATION BOARD

ON

THE ONTARIO GOLD MINING INDUSTRY

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4. STATEMENT OF DISSENT

Ontario
Ministry of
Labour

Ministère
du Travail
de l'Ontario

Industrial Disease
Standards Panel

Comité des normes
en matière de maladies
professionelles

10 King Street East
7th Floor
Toronto, Ontario
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416/965-5056

April 22, 1987

Dr. R. G. Elgie
Chairman
Workers' Compensation Board
2 Bloor Street East, 20th Floor
Toronto, Ontario
M4W 3C3

Dear Dr. Elgie:

In a letter dated August 18, 1986 the Board requested the Industrial Disease Standards Panel to respond to a number of questions about the occurrence of lung cancer and stomach cancer among persons who have worked in Ontario gold mines.

The Panel is pleased to send to the Board its Report of Findings in this matter in accordance with Section 86p(10) of the Workers' Compensation Act. While the Panel is unanimous with respect to both findings with respect to probable connection, there is appended a Statement of Dissent by two members of the Panel with respect to certain consequent issues.

In not finding a probable connection between stomach cancer and occupational groups within the Ontario gold mining industry, the Panel considers the evidentiary base as it now exists to be both biologically inconsistent with a premise of connection and inconclusive. The Panel intends to review the evidence when the Muller Report is updated and requests that as full as possible an update be carried out as soon as possible.

The Panel will be pleased to respond to questions the Board may have about its findings.

Yours sincerely,

James M. Ham

James M. Ham
Chairman

JMH:dp

Ontario Ministry of Labour
Ministère du Travail de l'Ontario

Industrial Disease Standards Panel
Comité des normes en matière de maladies professionelles

10 King Street East
7th Floor
Toronto, Ontario
M5C 1C3
416/965-5056

April 15, 1987

MEMORANDUM TO: Workers' Compensation Board

FROM: Industrial Disease Standards Panel

RE: **Report of Findings on Issues in the Ontario Gold Mining Industry**

The Panel is pleased to submit the attached Report of Findings on the issue of lung cancer and stomach cancer in the Ontario gold mining industry.

1. E. Gibson, Member
2. James M. Ham, Chairman
3. J. LeVay, Member
4. D.C.F. Muir, Vice Chairman

The following Members of the Industrial Disease Standards Panel offer the appended Statement of Dissent concerning the aforementioned issues of occupational disease in the Ontario gold mining industry.

J.L. Gagnon, Member

L. A. Jolley, Member

Ontario
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April 15, 1987

MEMORANDUM TO: WORKERS' COMPENSATION BOARD

FROM: INDUSTRIAL DISEASE STANDARDS PANEL

RE: **REPORT ON ONTARIO GOLD MINING INDUSTRY**

1.0 ISSUES AND FINDINGS:

1.1 In a letter dated August 18, 1986, the Workers' Compensation Board requested that the Industrial Disease Standards Panel provide to the Board responses to the following questions:

1. What is the Panel's opinion on the association between experience in Ontario gold mines and excess mortality from lung cancer?

2. What is the Panel's advice on the criteria to be considered for the compensation of gold miners who have subsequently developed lung cancer?

3. What criteria should the WCB employ to compensate mixed ore miners who have subsequently developed lung cancer?

4. Does there exist sufficient evidence for the WCB to provide compensation benefits to gold or mixed ore miners who have subsequently contracted stomach cancer?

5. If the answer to the fourth question is yes then what criteria should the Board employ to compensate this category of workers?

1.2 For reasons fully described in the Report of the Special Panel (attached hereto as Appendix A), the Panel believes that the original definitions of gold mining occupational groups as used in the Muller reports can be improved. The revised definitions as used by the Panel require exposure in "dusty" gold mining jobs (as set out in Table 5 of

the Report of the Special Panel) as the pivotal criterion for admission into one or more of the occupational cohorts used to study the Board's questions set out above. The Special Panel defined in particular an ALL GOLD MINERS cohort. As a direct consequence of this definition, the original separation into gold and mixed ore mining groups became no longer relevant. The Panel then set about responding to the Board's questions by examining the issue of occupational risk of lung and stomach cancers within its ALL GOLD MINERS (and related) groups.

1.3 The Panel has used as its evidentiary base the Muller et al. Reports (1983 and 1986), and supplements to the latter, as well as the Report of its own Special Panel on the Ontario gold mining industry and a further Report (authored by Miller et al.) prepared at the Board's request and subsequently provided to the Panel.

1.4 On the basis of this evidence:

FINDING 1: AT THIS TIME, THE PANEL FINDS A PROBABLE CONNECTION BETWEEN LUNG CANCER AND CERTAIN OCCUPATIONAL GROUPS WITHIN THE ONTARIO GOLD MINING INDUSTRY.

There is both sound statistical and biologically consistent evidence of a burden of excess lung cancer disease among these workers.

FINDING 2: AT THIS TIME, THE PANEL DOES NOT FIND A PROBABLE CONNECTION BETWEEN STOMACH CANCER AND OCCUPATIONAL GROUPS WITHIN THE ONTARIO GOLD MINING INDUSTRY.

There is not, in the evidentiary base used by the Panel, biologically consistent evidence of an occupational origin for excess stomach cancer among these workers.

1.5 The Board has, in a separate communication, requested advice on the matter of the 'healthy worker effect'. The Panel has not responded as yet on this issue and it is not considered further in this document.

1.6 The Panel urges the Board to update the Muller studies of the Ontario gold mining industry as quickly as possible and to make this update available to the Panel. The Panel will then review the new evidence to determine if any changes in these findings are in order.

2.0 THE BURDEN OF EXCESS LUNG CANCER DISEASE:

2.1 An estimate of the extent of the burden of excess lung cancer disease is given by the Standardized Mortality Ratios (SMR) derived by Dr. Muller, by Dr. Shannon (in the Report of the Special Panel), and by Drs. Miller et al., using a variety of cohort definitions. Dr. Muller's value for the lung cancer SMR is 140 and Dr. Shannon's values, for different cohort definitions, range from 130 to 143, while

Dr. Miller's estimate is 138. The cohort from the Special Panel's study that is deemed most relevant is the ALL GOLD MINERS cohort (see Table A below).

TABLE A

MULLER, SPECIAL PANEL AND MILLER COHORTS

Cohort	Person Years At Risk	Observed Deaths	Expected Deaths	SMR O/E	Excess O/E
Muller	111,265	165	117.5	140	47.5
All Gold Miners	150,181.5	209	148.5	141	60.5
Miller et al.	n/a	237	171.5	138	65.5

For admission to the ALL GOLD MINERS cohort, a person had to have at least five years of work experience in dust exposure in a gold mine either underground or in one or more of a particular set of surface jobs verified as "dusty" by the labour and management advisers to the Special Panel. Of the sixty months minimum, at least one half month had to be dusty work in an Ontario gold mine. For admission to the Muller cohort, a person had to have had 60 months of dusty mining experience including one half month in dusty Ontario gold mine work; and further, at least 85% of the person's mining experience had to have been in a gold mining environment. To be designated an underground gold miner in Muller's study, a person required at least 50% of his total work experience to have been underground in a gold mine. The Miller et al. study apparently includes all persons classified by Dr. Muller as mixed ore miners, some of whom have not had enough gold mining experience to qualify for his gold mining cohort or the Special Panel's ALL GOLD MINERS cohort.

2.2 With respect to gold mining experience, the Special Panel's ALL GOLD MINERS cohort is more specific than either the Muller or Miller et al. cohort. It is deemed appropriate to use this cohort in estimating the burden of excess disease carried by gold miners.

2.3 The reference value for further discussion of the SMR will be 141. Recall that the SMR is O/E in percent where O is the number of observed lung cancer deaths in the study cohort and E is the number of expected cases in the reference population. An SMR is a statistic which estimates an unknown "true" value. If an epidemiological study, as represented by the ALL GOLD MINERS cohort, were repeated in a number of situations like that of Ontario miners, different SMR's would be expected and, nineteen times out of twenty, these SMR's would be expected to lie between 123 and 161. These numbers are called the 95% confidence limits.

2.4 In the circumstances we are dealing with, we have no clear specific knowledge of the aetiology of an excess of lung cancer among gold miners. We know that in the atmosphere breathed by miners there have been historically varying amounts of silica bearing dust and of carcinogens such as arsenic and radon. The only quantified basis available to the Panel for estimating the burden of industrial disease is the epidemiological record. The epidemiological data, which reflect circumstances as assessed in the study period ending in 1977, provide a first estimate of the burden of excess disease as (O-E) where O is the number of observed deaths due to lung cancer (lung, bronchus, trachea) and E is the number of expected deaths in the reference population. For the ALL GOLD MINERS cohort in Table A, this number is 60.5. For the study period ending in 1977, this estimate is deemed to be conservative for several reasons discussed in the next two paragraphs. In addition, no cases of lung cancer of miners who died before 1955 or after 1977 are included in the study.

2.5 The Panel has structured the recommendations that follow with the intent of ensuring that the full extent of the burden of excess disease is compensated. Table A demonstrates that there are uncertainties in estimating the burden of excess disease that are associated with the definitions of different but related cohorts. Furthermore, with each individual study, there are associated bounds of uncertainty implied in the statistical methodology. It is more acceptable, when there is clear evidence of a burden of excess disease, to err on the side of over-estimation than of under-estimation. The issue is one of our society's respect for life, health and bodily integrity as fundamental values for those at work in a situation where the costs of compensation are collected from employers. While the circumstances that led to the burden of excess lung cancer among gold miners are believed for the most part to have characterized working conditions decades ago, the issue of compensation for this burden is to be judged today.

2.6 The Panel deems it appropriate to use the 95% rule as its policy basis for the true estimate of the burden of occupational disease. Simply stated, if the basis for asserting the existence of a "probable connection" is that the SMR must be significantly greater than 100 (in percent terms) at the 95% level, then, on the grounds of fairness, the upper bound on the estimate of the true value of the SMR should be used to determine the excess disease burden. Therefore, the burden of disease is defined as a number (O'-E), where E is the standard expected number of cases and O' is the number of observed cases that would correspond to the value of the SMR at the upper 95% confidence limit on the standard SMR. For the ALL GOLD MINERS cohort, E = 148.5, O = 209 and the standard SMR is 141. If the SMR is taken as SMR' = O'/E = 161, then O' = 239.3 and O'-E = 90.8.

2.7 **FINDING 3: ACCORDING TO THE FOREGOING PROCEDURE, THE PANEL'S ESTIMATE OF THE BURDEN OF EXCESS LUNG CANCER DISEASE IN THE ALL GOLD MINERS COHORT BETWEEN 1955 AND 1977 IS 91 CASES (90.8 ROUNDED).**

3.0 ELIGIBILITY RULES:

3.1 Explicit eligibility rules can be formulated only in terms of evidence available from that subset of the criteria of case adjudication which offer reasonably clear and biologically consistent indicators of the work-relatedness of the disease in question.

3.2 Dr. Muller's epidemiological study, the related studies by Dr. Miller et al. and by the Special Panel under Dr. Shannon show limited evidence of biologically consistent criteria of work-relatedness. These notes first review the Special Panel results and then comment on the Miller study.

3 Table 11 (Appendix A) shows that the standard SMR for lung cancer in the ALL GOLD MINERS cohort increases consistently from 67 to 159 as the time elapsed since first "dust exposure" increases from the interval 5-9 years to the interval more than 30 years. The longer intervals could only have been achieved if a significant part of such exposures took place before or during the second World War. Indeed the correlation desired in this regard is provided by Table 10 (Appendix A) which shows that the standard SMR for members of the ALL GOLD MINERS cohort whose first exposure was before 1936 is 165 and declines to 99 for those whose first exposure was in or after 1950. From the data of Table 10, the distribution of the Standard (O-E) estimate of excess deaths (of 60.5 in total) by time period is 50.3 for <1936, 11.3 for 1936-1944, 1.1 for 1945-1949 and negative for >1950. Thus almost all the burden of excess disease is attributable to the time period before 1945.

3.4 It is important to note from Table 9 (Appendix A) that there is no consistent evidence that increased duration of exposure (without regard to when it occurred) leads to increased risk of disease.

3.5 The foregoing evidence is consistent with the fact that the "dustiness" of mine atmospheres may be deemed to have declined to present day levels from much more severe historical levels. This is confirmed by information from the Report of the Royal Commission on the Health and Safety of Workers in Mines (1976, p. 44), from records of historical dust levels provided through the Ontario Mining Association (Appendix B, Muller, J., letter dated June 13, 1985) and from advice provided by labour and management advisers to the Special Panel (Report of the Special Panel, Section 1.2, p. 16-18).

3.6 The Special Panel analyzed the SMR for lung cancer as a function of duration of exposure weighted by a dust index indicating dust severity by calendar period (and defined in Appendix A, p. 25-26). The resulting data are shown in Figure 2 (Appendix A) where Excess Relative Risk = SMR-1 = (O-E)/E is plotted versus weighted years of dust exposure. Figure 2 provides a relatively linear dose-response function which is highly pertinent to the issue of compensation.

3.7 Using Figure 2 as a dose-response function, it may be judged that cases of lung cancer for which the weighted dust exposure index is highest are most likely to represent the excess disease burden among the mining occupations exposed to gold dust. Accordingly it is proposed that the following eligibility rule be adopted by the Board:

ELIGIBILITY RULE 1: THAT LUNG CANCER CASES AMONG ONTARIO GOLD MINERS IN DUSTY OCCUPATIONS (APPENDIX A, TABLE 5) WHOSE WEIGHTED DUST EXPOSURE IS 60 OR MORE BE COMPENSATED.

This would have the effect of compensating those cases of lung cancer with 15 years of exposure prior to 1936, with 17 years prior to 1945, or with 22 years of exposure prior to 1955.

3.8 From Table 15 (Appendix A), the foregoing rule would lead to the compensation of 82 cases which would represent 90 percent of the estimated burden of excess disease within the perspective of the ALL GOLD MINERS cohort for the period 1955-1977 which includes dust-exposed occupations as listed in Table 5.

3.9 The panel makes the following further recommendations:

RECOMMENDATION 1: THAT THE BOARD METICULOUSLY SEARCH OUT ALL HISTORICAL CASES OF LUNG CANCER AMONG GOLD MINERS;

RECOMMENDATION 2: THAT FOR ALL CASES ELIGIBLE FOR ADMISSION TO AN HISTORICALLY EXTENDED ALL GOLD MINERS COHORT, ELIGIBILITY RULE 1 SHALL APPLY. MOREOVER, A CAREFUL CASE ASSESSMENT MUST BE CARRIED OUT USING THE TOTALITY OF INFORMATION THAT CAN REASONABLY BE COLLECTED WITH RESPECT TO ALL CRITERIA OF CASE EVALUATION CITED IN SECTION 4.0;

RECOMMENDATION 3: THAT ALL OTHER CASES BE ADJUDICATED ON THEIR MERITS. THE PANEL NOTES IN THIS REGARD THAT IT IS COMMON FOR A MINER IN THE COURSE OF HIS CAREER TO WORK IN A VARIETY OF OCCUPATIONS AND IN A NUMBER OF MINES. WHILE THESE CIRCUMSTANCES CREATE A MEASURE OF AVERAGING OF CONDITIONS OF EXPOSURE IT IS CLEARLY POSSIBLE FOR INDIVIDUALS TO HAVE EXPERIENCED EXPOSURES OVER TIME MORE INTENSE THAN ARE INDICATED BY WEIGHTED AVERAGE DUST EXPOSURE;

RECOMMENDATION 4: **THAT IN EFFECTING CASE ASSESSMENTS, THE BOARD DRAW FROM ALL THE CIRCUMSTANCES OF THE CASE AND ALL THE EVIDENCE DISCOVERED BY OR PRESENTED TO IT EVERY REASONABLE INFERENCE IN FAVOUR OF THAT CLAIMANT; AND ACCEPT AS PROOF OF ANY FACT THAT THE CLAIMANT IS REQUIRED TO PROVE, ANY CREDIBLE EVIDENCE SUBMITTED BY HIM THAT IS NOT CONTRADICTED AND WHERE, IN WEIGHING ANY EVIDENCE SUBMITTED TO IT, ANY DOUBT EXISTS AS TO WHETHER THE CLAIMANT HAS ESTABLISHED HIS CASE, THE BOARD SHALL RESOLVE SUCH DOUBT IN FAVOUR OF THE CLAIMANT (DRAWN FROM THE REPORT OF THE ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO, CHAP. 13, SECT. D, BENEFIT OF DOUBT POLICY, PP. 765-7).**

4.0 CRITERIA OF CASE EVALUATION:

4.1 It is impossible to identify with complete assurance the particular subset of all observed cases of lung cancer that represent the burden of excess disease. The extent of the global burden of disease can, as we have just seen, be at best estimated. Nevertheless, in the situation at issue, there is an excellent data base capable of yielding statistically strong global estimates.

4.2 To deal with the question of criteria of evaluation we must conceive of assessing each and every observed case according to a set of criteria and then of ranking all the cases in descending order of likelihood of being work related (and therefore part of the burden of excess disease) according to the "weight" given to the various criteria of judgement. If we imagine this having been done, and having in mind the total number of cases that represents the estimate of the burden of excess disease, we then partition off a number of cases approximately equal to the estimated global burden of excess disease. The rules for doing this first partition are considered by the Panel to constitute eligibility rules. These rules are to be interpreted basically as being rules of presumption of compensability. These rules are to be derived from the structure of the available evidence which in our situation is largely epidemiological. Such rules do not preclude the adjudication of any case claim on its merits in relation to all available criteria of evaluation.

4.3 In the process of judging individual cases, "the benefit of the doubt" is to apply within the overall context of a defined global burden of excess disease and the adjudicatory process of weighing evidence.

4.4 With respect to claims by miners, the following are 12 generic criteria within which a case claim may be assessed. The specificity of available information for individual cases or groups of cases under the different criteria will vary enormously:

4.4.1 Geology

The nature of the valuable mineral in the mine together with the configuration of the deposit (veins) and the nature of the associated matrix of waste rocks and trace elements.

Comment

This criteria can be used to distinguish one mine from another. Uranium is ubiquitous in the earth's crust and therefore radon and thoron gas and gamma rays, will be emanated at associated levels. Arsenic and fibrous substances may be present at varying levels in minerals. Different levels of silica characterize ore bodies.

4.4.2 Mine Design

The configuration of the mine in terms of shafts, adits, drifts, stopes, drawholes, and transportation practices has a bearing on the potential of the mine to be associated with risks to health in its working atmosphere.

4.4.3 Mine Operation - Technology

Associated with the mine configuration is the kind of technology used to extract ore. This technology relates to: i) drilling; ii) blasting; iii) transporting broken rocks: iv) crushing; v) water supply; vi) ventilation; vii) grinding: and viii) separation processes.

Comment

Some mines are of large scale and use (for example) diesel-operated vehicles which produce fumes. Others are of small scale. In any event the technology of a mine may undergo significant changes over the lifetime of the mine and have significant influence on the risks to health expressed in the working atmosphere.

4.4.4 Mine Operation - Work Practices

Mine design and technology largely determine the categories and numbers of jobs associated with company operations. It is useful to identify the relative exposures likely to be associated with specific jobs.

4.4.5 Mine Operation - Production

A record of production levels is pertinent to the levels of exposure that may be expected in various calendar periods.

4.4.6 Mine Operation - Mine Environment

With respect to lung disease, the character of the mine environment that workers may encounter is of particular concern. All evidence in records related to total dust, silica, radon and other sources of radioactivity, diesel fumes, arsenic, carbon monoxide, mists from drilling, from metallurgical separation processes etc., may be of relevance. Historical records are often missing and trends for conditions in particular mines may only be capable of being inferred from historical industry trends and competent judgments of the "quality" of particular mines.

4.4.7 Work History - Occupations

The particular occupation (job category) or occupations carried out for specific calendar periods in specific mines represent key information relevant to a case claim. The significance of these data is to be seen in the context of the preceding criteria.

4.4.8 Work History - Year of Start Mining

Since environmental conditions are known to have changed significantly from the early days of mining in Ontario to the present, the starting point for a working career in mining is of particular interest.

4.4.9 Work History - Age at Start of Mining

Initial age may be an important variable in conjunction with items 8 and 10 related to the likelihood of the onset of industrial disease.

4.4.10 Work History - Years in Mining and Other Occupations

The sequence of years spent by a worker in the various occupations in various mines and other occupations which describe his working lifetime is information of particular relevance to a case claim.

4.4.11 Personal Medical History

Available information (concerning chest x-rays, other medical diagnostic records and work-related characteristics) is critical to case assessment and may provide, as in the case of chest x-rays, surrogate evidence of the dustiness of mine atmospheres.

4.4.12 Other Worker Populations

Evidence for other working populations in other jurisdictions may be useful.

5.0 COMMENTS ON MILLER ET AL. REVIEW OF EVIDENCE:

5.1 The Board commissioned Miller et al. to provide advice on the issue of compensation among gold miners for lung cancer and stomach cancer. It was initiated only after the Panel advised the Board in December, 1987, that it was unable to provide advice to the Board in the short time frame the public circumstances were deemed to warrant. Nonetheless, the Special Panel study by the Panel and the Board's separately commissioned study have been completed at the same time and therefore represent independent examinations of the same issues.

5.2 Miller et al. confirmed the appropriateness of Muller's methodology, and of his conclusion that gold mining, as an occupation, has shown a clear association with an increased risk for lung cancer. Since they found that the lung cancer experience for mixed ore miners was similar to that for gold miners, they have not distinguished between the two groups in their recommendations for compensation.

5.3 One of the major concerns expressed in the Miller et al. Report in attempting to establish eligibility rules for lung cancer was the fact that Muller's data did not show a clear relationship between excess lung cancer and duration of exposure or between excess lung cancer and time since first exposure. In the absence of such a clear relationship they considered radiological evidence of silicosis to be a reasonable marker of "dose". They were able to identify two subgroups which approximated the excess burden of lung cancer: miners employed before 1945 with chest x-ray rating B (Ontario Code 4) or C (Ontario Code greater than 4).

5.4 The use of a chest x-ray rating as a decision criterion poses a clinical/diagnostic problem. While epidemiologically appropriate for identifying groups at risk, there are potential biases when applied to individuals. There is well recognized inter- and intra-observer variation in assigning categories of rating. It would be more likely for an individual with a previous positive reading to be assigned to a positive category (even if the "truth" were negative), and the key decision, based on when, for example, a Code 3 becomes a Code 4, increases the likelihood of assignment error.

5.5 There is also concern about the premise that the presence of silicosis would identify increased risk for lung cancer because silica is a carcinogen. Although some studies suggest that silica may be a carcinogen, the issue remains controversial, and the International Agency for Research in Cancer (IARC) has categorized silica as having "limited" evidence for human carcinogenicity.

5.6 In the Report of the Special Panel (Section 2.1), the lack of a strong association between the excess silicotic cases removed from the 1986 gold mining cohort and lung cancer weakens the argument of silicosis being a primary cause of lung cancer. Further, foundry studies have shown an excess risk for lung cancer similar in magnitude to that for gold miners but a much smaller incidence of silicosis. These discrepancies cast doubt on the role of silica as a primary carcinogen in gold miners, and it would be inappropriate to use silicosis as a "marker".

5.7 Miller et al. outlined several difficulties with the excess stomach cancer cases that precluded their acceptance as being causally related to gold mining. Among the concerns were: an over-representation of men not born in North America; a reversal of a possible dose-response relationship (i.e. only a slight increase in mortality in those with the highest dust exposure, and a significant excess in those with the least exposure); the finding of no increase in stomach cancer in "mixed ore" miners as compared to "gold" miners, while both have similar risks of excess lung cancer; and a marked deficit in other gastro-intestinal cancer.

APPENDIX A

REPORT OF THE SPECIAL PANEL

ON

ONTARIO GOLD MINING INDUSTRY

REPORT OF THE SPECIAL PANEL ON THE ONTARIO GOLD MINING INDUSTRY

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FOREWORD

The Special Panel was chaired by Dr. Harry Shannon, Associate Professor, Occupational Health Program and Department of Clinical Epidemiology and Biostatistics, McMaster University. He was assisted in his investigation by the following staff of the Industrial Disease Standards Panel: Dr. James G. Heller, Executive Administrator: Ms. Gaylene Pron, Senior Research Officer; Mr. Paul Gallina, Research Officer: and Mr. James MacRae, Research Officer.

ACKNOWLEDGEMENTS

The Special Panel is indebted to many people for their direct and indirect assistance in preparing this report.

The Mining Master File of the Workers' Compensation Board is a remarkable and unique data source. We are grateful to its compilers and to the Board for the use of the information it contains.

We thank Dr. Jan Muller, Bob Kusiak and George Suranyi - authors of the original reports on Ontario miners - for spending time with us to discuss their studies and to make available additional data. Indeed, this report and its analyses have relied on and benefited from the pioneering efforts of Dr. Muller and his colleagues. We wish to acknowledge the help of Prof. R.W. Thompkins of Queen's University and Mr. Gordon Prest of the United Steelworkers of America, who explained to us much about the history and practices of the Ontario gold mining industry.

The Systems Branch of the Ministry of Labour was extremely generous and responsive to our request for computer support. In particular, Annette Matuszek contributed her excellent scientific programming skills to the project and she and her supervisor, Mr. James Tse. are to be commended.

Dr. Aileen Clarke, Dr. Lorraine Marrett, Darlene Dale and Donna Reynolds of the Ontario Cancer Treatment and Research Foundation assisted the Special Panel in checking the diagnoses of the cancer cases among the miners.

Several mining companies co-operated in the validity checks we conducted: Dome Mines, Campbell Red Lake and Kerr-Addison. The United Steelworkers of America also supplied useful information. Mr. Bruce Campbell of the Ontario Mining Association provided additional material.

Mrs. Lois Bouskill and Mrs. Dorothy Plummer typed sections of the document.

EXECUTIVE SUMMARY

1. The aim of this report was to examine the evidence that gold mining had led to an increase in lung and stomach cancer in miners. This concern followed two reports of Ontario miners which showed more deaths than expected from these diseases.

2. A review of the literature on gold miners elsewhere in the world found that there was generally an increase in lung cancer in this occupation, while the data for stomach cancer were inconclusive.

3. Advisers to the Panel described past practices in Ontario gold mines. Discussions focussed on environmental contaminants and changes in hygiene conditions over time.

4. Validity checks were conducted on the completeness and accuracy of the Mining Master File, which was used in the analyses. The information was found to be of very high quality.

5. Death certificate information was checked at the Ontario Cancer Registry using all information available. The diagnoses of the cancers were found to be of comparable quality to those for the Ontario male population.

6. Further analyses were conducted using a re-defined cohort. This included all men who worked in dust exposure in gold mines for a total of five years or more, of which at least half a month was in an Ontario mine between 1955 and 1977. A miner was excluded if he was known to have had previous exposure to asbestos or in uranium mining or processing. If such exposure started after his gold mining, person-years were calculated up to the date the exposure began.

7. The overall mortality risks from various causes were very similar to those found in the previous reports. In particular, deaths due to lung cancer and stomach cancer were both significantly increased, about 40% greater than expected.

8. Detailed analysis of the lung cancer mortality showed that the Standardized Mortality Ratio (SMR), a ratio of observed deaths to the number expected based on the general population, increased with time since first exposure and was greater the earlier the first dust exposure in gold mines.

9. An index of weighted exposure was constructed to take into account the reduction in dust levels over time. It was intended to provide a measure of cumulative dust exposure. A clear and consistent dose-response relationship was found, the SMR's increasing with increasing weighted exposure. It was concluded that the increase in lung cancer was consistent with an occupational cause.

10. The lack of information on specific contaminants makes it impossible to establish the likely cause of the excess lung cancers.

11. A ranking of the likelihood that any case of lung cancer was caused by work in gold mines can be derived from the weighted exposure index- the higher the value of the index, the greater the probability.

12. The detailed analyses for stomach cancer showed inverse dose-response relationships - the opposite patterns to those expected with an occupational cause. On the basis of the evidence, one cannot conclude that the increase in stomach cancer has been occupationally caused by exposure in gold mining.

SECTION 1

OVERVIEW

1.1 ISSUES:

In 1983 the "Study of Mortality of Ontario Miners: 1955-1977" was issued by Dr. J. Muller and his colleagues (1). Several findings of disease risk were noted among different Ontario mining groups. In particular, there was an increase in lung cancers among uranium, gold and mixed ore miners and there was an increase in stomach cancers among gold miners. The gold mining cohort was studied in more detail and in 1986 a further report was issued (2). The 1986 report addressed both the issues of increased lung and stomach cancers among gold miners. Two supplements to this latter report provided further information on lung and stomach cancers for both gold and mixed ore mining (3).

These studies were generally well conducted. Nevertheless, a number of criticisms concerning them have been raised and the studies have been reviewed in some detail. Moreover, additional analyses have been conducted to examine further the excess cancers among gold miners. The purpose of this overview is to discuss some of the criticisms and to provide a rationale for the extra analyses that have been conducted.

The original (1983) definition of the gold mining cohort was as follows. The Mining Master File contained information on all men with at least 60 months of mining experience (of which at least one half month had been in an Ontario mine) and who had been x-rayed at an Ontario Miners' annual examination at some point since 1955. Men with known asbestos exposure or who had previously been employed in a uranium mine or processing plant were excluded. In the second (1986) report the definition was modified. Those men no longer working in a mine during the study period (1955-77) even though they continued to be x-rayed, were omitted, since they might be self-selected for attendance at miners' examinations. This modification was

appropriate, since any bias would likely be related to health status. A further criterion was necessary to place a man in the "gold mining" category as opposed to the "mixed ore" group. This criterion took each miner's total experience up to the end of the study period and placed him in the ore cohort corresponding to the "85% rule". To be a gold miner meant that 85% or more of a man's mining experience was in a gold mine. A difficulty with this definition is that the categories were established a posteriori. For example, an individual with twenty years of gold mining experience when he left the industry would have had all his person years up to that date (and subsequently) allocated to the gold mining cohort. He may have worked side by side with another miner during those twenty years, but the second miner may have moved to a nickel/copper mine and worked an additional ten years. If this second man then left the industry, all of his person years (including his gold mining) would be attributable to mixed ore mining using the study definitions. Thus, the person years for two men working side by side during the period in which their experience up to that time had been identical would be allocated to different sub-cohorts. This is inappropriate, although when the study was originally planned, no other method seemed more reasonable. However, the results from the 1983 study and other studies revealed no apparent cancer risk in miners of other ores (apart from uranium). For this reason, an alternative definition of a "gold miner" was developed and used in the analysis below.

Data quantifying the levels of contaminants to which gold miners were exposed are not available. This clearly limits any inferences concerning environmental agents and makes a proper approach to the analysis more difficult. From all accounts (including both verbal comments from mining experts and a review of ventilation practices), conditions have significantly improved over the last forty or fifty years. In addition, the attention to work practices and blasting methods have reduced dust exposure for miners. Apparently, however, changes have varied from mine to mine so that while many improvements began to occur in 1945, they did not occur suddenly but took place over a period of a few years. By taking account of the dates when men first worked, and the length of time they worked, some indication of the amount of exposure received can be obtained.

Mortality was determined through record linkage with the National Mortality Data Base held by Statistics Canada. This method will generally tend to under-estimate mortality. A check with records of men known to have died (pensioners at INCO also in the cohort) found that 94% had been correctly linked with the mortality data base. This approach, however, may slightly over-estimate the quality of the linkage, since it was conducted on pensioners for whom company records will be very good because of their long service with the company. If longer term workers are more likely to be linked because of better quality information in the Mining Master File, then any dose-response relationship will be slightly accentuated. On the other hand, this will probably be counter balanced by a tendency for earlier records

(mainly of men with longer exposure) to contain poor information and therefore be less likely to be found through the record linkage procedure. Overall, any errors in determining deaths will probably have had only a small effect on the conclusions of the study, although the point estimates of any risks will have been reduced.

A potentially more serious problem is the possible effect of diagnostic errors. It was pointed out that lung cancer might be diagnosed more often (and erroneously so) among gold miners because they had frequent x-rays due to the greater incidence of silicosis and silicotuberculosis. Subsequent reports though, have noted that histological confirmation of lung cancer have been obtained in 87% of the 165 observed cases and in only three of the observed cases was silicosis also diagnosed. On the other hand, it could be that lung cancer is under-diagnosed in the general population, thus leading to an under-estimate of the expected number of such deaths in the gold mining cohort. The problem of diagnosis may also have applied to stomach cancer. It has been argued that, since the excess of stomach cancers was roughly the same as (indeed, less than) the deficit in cancers at other sites of the digestive system, the raised SMR for stomach cancer may have been an artifact. Muller and his colleagues did not obtain histological confirmation for the stomach cancer cases.

A further difficult question is whether silica exposure, silicosis and/or silicotuberculosis are precursors to lung cancer or are simply associated with it by virtue of heavy exposure. That is, death due to silicosis or silicotuberculosis may reflect high exposures not only to silica but also to other contaminants among which may be a lung carcinogen. It is probably impossible to sort out this question given the crudeness of the information available on environmental contaminants.

In this respect though, it is interesting to note the effect of the different cohort definitions used by Muller and his colleagues. The 1983 study included men no longer working in mines, and this sub-group was omitted from the 1986 report. Among these men, the excess lung cancer (observed deaths minus expected) was 13.6, while the number of cases of silicotuberculosis, silicosis and chronic interstitial pneumonia was 99. The corresponding numbers for the men included in the 1986 cohort were 47.5 and 53. If silica, which causes silicosis, were also the major cause of the lung cancer increase, one would have expected a closer relationship between the ratio of excess lung cancer to silicotic diseases. However, the selection factors in the men omitted from the second report make it impossible to determine just how close the two ratios should be.

It is always difficult in this type of study to know the appropriate control group for comparison with the occupational group under study. As is common practice, we will follow Muller et al. and use the Ontario male population.

Two points have been raised: firstly that the rates in Northern Ontario of lung and stomach cancer are higher than in the rest of the Province and, secondly, that stomach cancer rates among immigrants (who form a high proportion of the miners) are higher than in the rest of the provincial population. The latter point is probably reasonable, but the first creates some difficulty. Many of the Northern Ontario towns are mining towns, and if there is any effect of mining on lung cancer, then comparison with the Northern Ontario population may result in an over-adjustment if miners are a significant proportion of the total males employed there. However, a dose-response relationship will be little affected by the choice of comparison group.

A case-control study was also conducted among the lung cancer cases. In this type of study, cases of a particular disease (in this case lung cancer) are compared with a matched group who do not have the disease in terms of their previous exposure. The advantage of this type of study is that with the smaller numbers involved it is possible to obtain more detailed information on a variety of factors which might have affected the results. Thus, it may be possible to determine whether smoking habits may have accounted for at least some of the increase in lung cancer among the gold miners.

By their nature, case-control studies have limitations. In this study, for example, information on smoking could not be obtained for all cases of lung cancer or all controls selected. It is impossible to know in which direction any bias caused by this lack of information may have occurred; that is, whether the missing information may have led to an under-estimate or over-estimate of the effect of gold mining. Such data as were available, though, suggest that smoking was not responsible for the observed increase in lung cancer. The amount of smoking was very similar to that in the nickel/copper miners among whom no significant increase in lung cancer was observed. In addition, none of the analyses in the case-control study contradicted the hypothesis that lung cancer was related to gold mining.

1.2 MINING PRACTICE IN ONTARIO

The mining industry forms an important part of Ontario's economy. Mineral production in Ontario is 30 percent of the Canadian total, if fuels are excluded. Gold production in Ontario is just slightly above this national average with approximately 33 per cent of the Canadian total. Globally, the Soviet Union and South Africa account for over 80 per cent of the world's annual production from over 60 countries. Canada's share of this market is approximately 3 per cent.

Over the years gold production in Ontario has varied greatly given changing economic conditions and mine practices. In 1984, for instance, 26.5 metric tons, valued at $399.7 million was extracted compared with 76.6 metric tons in 1957, worth $91.1 million. Ontario gold production from 1935 to 1985 and its dollar value (in 1971 constant dollars) are shown in Figure 1.

To provide some historical and technical insights about the working conditions in Ontario gold mines, meetings were held at Panel headquarters with advisers Professor R.W. Thompkins (nominated by the Ontario Mining Association) and Mr. Gordon Prest (nominated by the United Steelworkers of America). An additional objective of the meetings was to provide some independent verification of the assumptions made by the Workers' Compensation Board (WCB) in its coding practices for job titles used to determine dust exposure.

There was a discussion of some of the types of contaminants that occur in gold mining environments: arsenic, silica, dust, asbestos, radon, diesel fumes, carbon monoxide, sulphides and methane gas. It was noted that arsenic is a contaminant in both gold and other types of ore. In Ontario gold mines, arsenic occurs in varying concentrations in the different geographic regions. Mr. Prest identified gold mines in the Red Lake area as having high levels of arsenic contamination. Although neither of the advisers was aware of any studies to determine the presence of asbestos in gold mines, there was consensus that the only likely asbestos exposure would have arisen from its use as insulation. The introduction of diesel-driven technologies occurred during the sixties, so that diesel fume exposure is a fairly recent phenomenon. No survey on radon exposure in non-uranium mines was identified during discussions. Additional toxic exposures such as mercury and cyanide were identified by Mr. Prest as occurring only during ore processing.

Changes which have occurred over the years in ventilation and other dust suppression practices provided a second major focus of the meetings. A strong distinction was made by Mr. Prest between changes that occurred in gold mining versus those in other base metal industries. Specifically, gold mining was often conducted on a smaller scale and in a more labour-intensive fashion than other ore mining because the ore body often could not support bulk mining techniques. Also the tighter profit margins for the smaller mines may have been a factor in their ability to introduce technological changes, such as improvements in ventilation. Thus, there were often variations in ventilation and other mining practices between larger and smaller operations, with working conditions in the latter generally being poorer.

Both advisers agreed that significant reductions in mining dust levels have occurred due to changing practices. However, such changes were introduced unevenly in the various mines. Some of the mentioned advances were: drilling practices (i.e. burn cut versus V cut); the use of clay rather than wood spacers; vented drill heads; the use of atomizers; re-entry restrictions after blasting; and generally improved ventilation techniques. Professor Thompkins gave a general description of mine ventilation techniques. It was agreed by both advisers that during mine development it is not always possible to have well ventilated areas.

The advisers were asked about the classification of job titles into exposure categories. Various job titles were selected because they were atypical and not part of the Workers' Compensation Board coding manual. These job titles were subsequently sent to the WCB to determine their coding practices concerning dust exposures. When the WCB's practice and the advisers' assessments were compared, there was very good agreement on the opportunity for dust exposure for different job categories.

1.3 THE LITERATURE ON MORTALITY AMONG GOLD MINERS FROM LUNG AND STOMACH CANCER

The mortality experience of miners has been investigated in various hard rock mining industries and increased lung cancer mortality has been documented for miners in fluorspar (4) and in various metal mining industries, notably tin (5), iron (6,7) and uranium (8,9). There have also been various studies investigating the mortality experience specifically of gold miners. The results of these largely historical cohort studies, in 5 different countries - Australia (10), South Africa (11), France (12), United States (13, 14) and the Soviet Union (15) - will be discussed in the following review. The studies will be compared in terms of methodologies, observed mortality risks, dose-response relationships of risk and effects of potential confounding factors.

Lung Cancer Mortality

Lung cancer risk estimates and associated confidence intervals are summarized in Table 1 for the five cohort studies. A major methodological difference between the studies was the method of selecting eligible miners for the studies. The Soviet and both American studies selected eligible miners from a particular mining company on the basis of company employment records. The South African and the Australian studies selected eligible miners from mining regions on the basis of information derived from compulsory annual health examinations. Despite this and other differences in study methodologies and gold mining practices in the compared countries, a fairly consistent mortality pattern was observed.

All but one of the studies (13) found a significantly increased risk using the standardized mortality ratio (SMR) of lung cancer in gold miners. In the Soviet study, the relative risk (RR) was estimated, which, under certain assumptions, is equivalent to the standardized mortality ratio. The increased risks for underground gold miners in the various cohort studies were as follows: in the Australian cohort of 1,974 miners followed for 14 years the SMR was 140; in the South African cohort of 3,971 white miners followed for 9 years the SMR was 161; in one American cohort (Lead, South Dakota) followed for 14 years the SMR was 370; and in the Soviet cohort followed for 27 years the RR was 7.9. The study on 1000 French miners followed for 11 years in the Salsigne gold mining complex reported 66 bronchopulmonary cancer

deaths with a resultant SMR of 1034. However, the large SMR estimate of this study appears to be an anomaly compared to the other study estimates and interpretation of these study results is further complicated by the uncertain description of eligibility criteria for miners in the cohort. The other cohort study of American gold miners from the Homestake mining company also conducted in South Dakota did not report an increased risk of lung cancer (SMR = 100). The Homestake cohort was larger (3,328 miners) and was followed for a longer period of time (26 years) than the Lead cohort of gold miners. However, the expected number of lung cancer cases for the Homestake cohort was derived from National rather than State mortality rates used in the Lead study. The authors note that National lung cancer rates were higher than South Dakota rates where the majority of the miners resided; and if State rates had been used, the SMR for lung cancer would have been elevated by 38 % to 138.

Stomach Cancer Mortality

The pattern for stomach cancer mortality for gold miners based on these cohort studies was inconclusive. Stomach cancer mortality was not examined in either the French study or the American (Lead, South Dakota) study of gold miners, perhaps indicating that there was no observed increase in this disease. Stomach cancer risk was reduced in both the Australian (SMR = 40) and the American Homestake, South Dakota (SMR = 57) cohorts of underground gold miners. Evidence of an increased stomach cancer mortality for the South African cohort of white gold miners (SMR = 157) was, however, not statistically significant. However, an increased risk of stomach cancer was observed in the Soviet study for underground (RR = 5.7) but not for surface workers (RR = 1.1). Hence this review has identified two studies (11, 15) outside Canada which have found an excess of stomach cancer mortality in gold miners (although only one (15) was statistically significant) and two showing a deficit for the disease.

Causal Associations between Lung and Stomach Cancer Mortality Risk and Gold Mining Experience

Increased risks of mortality from lung cancer reported for underground gold miners in the above retrospective cohort studies may be attributable to mining or non-mining experiences. One method to check on the occupational link is to see if mortality risk increases with increasing amounts of mining exposure, thus supporting a finding of a causal or 'true' association. This review has therefore examined the studies for any such relationship. We should be aware, though, that in gold mining as in any hardrock mining, determinations of "exposures" are complicated by the possible presence of different disease-inducing agents, by changes in conditions over time, and by inadequate historical or current information on exposures.

Attempts to establish dose-response relationships for lung cancer were made in all but the French study (no such analyses were conducted for stomach cancer). " Exposure" was generally represented by years of underground mining or years of work in dust exposure which, given changes over time, may not be an accurate measure of actual exposure.

The mine environments in the different countries for the gold mining cohorts varied, and years of mining service were likely to represent different exposures. Arsenic was reported by the authors to be very high in France, to be at trace or background levels in the American and the Australian mines and to be absent in the South African mines. Asbestos (noncommercial forms of amosite) levels in both South Dakota mines were reported to be at about occupational standards (2.0 fibers /cc) recommended by the National Institute for Occupational Safety and Health, and levels of asbestos were not mentioned for the mining regions in the other cohort studies. Silica levels were mentioned in the two American cohort studies: there was 39 % free silica in both the Lead and Homestake South Dakota gold mines. Radon and/or radon daughter levels were reported in all of the studies to be low and below the currently regulated standards of 4 WLM per year.

Dose-response relationships in the Soviet Union study were loosely examined by comparing lung and stomach cancer mortality for underground and surface subcohorts of gold miners. For both lung (RR = 7.9 vs RR = 1.6) and stomach cancer (RR = 5.7 vs RR = 1.1) the relative risk was significantly increased for underground but not for surface workers.

In the American study of Lead gold miners, risks of malignant and nonmalignant respiratory diseases were compared between two groups of miners, one with 5 to 19 years of underground service and the other with 20 or more years of underground service. For nonmalignant respiratory diseases and for pneumoconiotic disease specifically, a significant excess of mortality was observed only with the longer exposure group. However, for malignant respiratory disease, there was a significantly increased risk in both groups of miners.

The other (larger) American study at Homestake which showed no lung cancer risk for underground gold miners also showed no linear trend of increasing risk with increasing duration of underground employment, latency or cumulative dust exposure. However, lung cancer risk did increase with increasing length of time underground from time periods of 1 - 9, to 10 - 14 years, and to 15 - 19 years, but levelled off after 20 years of employment.

In the Australian study attempts were made to establish a dose-response relationship using two different measures: surface versus underground groups of miners: and years of underground employment. Although underground miners had a relative risk of 1.41 for lung cancer compared to surface miners, it was not statistically significant (95% Confidence Interval about the risk = 0.69 - 2.96).

Again no linear trend was observed for age standardized lung cancer mortality rates with increased duration of underground mining. However, the lung cancer mortality rate did increase with increasing duration of underground mining from none to 1 - 19 years, then levelled out at 20 years. This pattern of lung cancer risk, increasing with increasing durations of underground mining and then levelling off, was the same as that in the Homestake miners.

The Australian study was one of two to examine the effects of cigarette smoking and mining experience on lung cancer risk. Information on smoking habits was obtained from the miners in a survey conducted at the beginning of the study. The highest lung cancer mortality rate was observed among miners who smoked and worked underground (3.60 per 1000 person-years) whereas a lower rate was observed among those who smoked and worked on the surface (2.62 per 1000 person years). The relative risk (RR) of lung cancer for these two groups of miners was 1.39, but this was not statistically significant (95% Confidence Interval about the RR was 0.68 - 2.93).

A more detailed examination of dose-response was conducted for the South African gold miners. A case-control study was used to examine further the relationship of lung cancer risk (RR) with mining experiences (dose measures based on duration of underground service and cumulative dust exposure) and personal characteristics (smoking frequency and systolic blood pressure) based on information obtained from compulsory annual medical examinations. The greatest risk factor for lung cancer and for chronic respiratory disease, based on estimates by logistic regression, was cigarette smoking i.e., RR=3.74 and 3.67 per 20 cigarettes smoked daily. The estimate of lung cancer risk with cumulative dust exposure (after controlling for smoking and systolic blood pressure) was elevated (R.R=1.77), although not significantly so (95% C.I. = 0.94 - 3.31), whereas with years of underground service, no increased risk was found (R.R = 1.14, 95 % C.I. = 0.63 - 2.05).

Conclusion

In summary, there appears to be adequate evidence from the cohort studies reviewed in this paper to support a finding of increased mortality risk for lung cancer in gold miners. No studies were able to determine a linear dose-response relationship between years of mining service and lung cancer mortality although increasing risk was observed for limited ranges of employment years. The evidence for stomach cancer mortality in gold miners is more difficult to interpret than that for lung cancer. Results are equivocal with some studies showing an increase in the disease, with others finding a deficit. Differences in the types and levels of exposure might explain this, but appropriate data are not available. Overall, the relationship between gold mining and stomach cancer remains inconclusive.

SECTION 2

VALIDITY OF THE MINING MASTER FILE AND DEATH RECORD CERTIFICATION

2.1 VALIDITY OF THE MINING MASTER FILE

The validity of using the Mining Master File (MMF) to capture the employment experience of Ontario gold miners was assessed in terms of its inclusiveness in identifying eligible miners and its accuracy when compared with mining company employment records.

The eligible study population in Dr. Muller's 'Study of Mortality of Ontario Gold Miners: 1955-1977' was defined as all miners:

1. Employed at any time between January 1, 1955 and December 31, 1977 (as determined by the MMF record of their radiological examination);

2. With at least 60 months of gold dust exposure of which at least 0.5 months was underground in Ontario gold mines;

3. With no previous exposure to asbestos or uranium mining and milling or at Eldorado operations.

The inclusiveness test of the MMF was undertaken using employment records obtained from three gold mining companies located in the three principal gold mining districts in Northern Ontario. The company records were randomly sampled to obtain a list of 288 miners which was then sorted by name and miner certificate number. Data on these men were compared with information on the MMF. Three miners from the company list were not found on the Mining Master File (giving an inclusion rate of 99%).

The validity of employment histories on the MMF (as reported by miners at their annual x-ray examinations) was tested by comparing them with company payroll records. The miners' self-reported histories included: mining company name, job type and duration of employment for each job held since their last x-ray examination. For a random sample of 100 miners from the list of 285 matched miners, comparisons were made for agreement on the date each miner started working at that mine and on the duration of employment. Records for five men were omitted because early company records were missing.

The test for agreement on a miner's starting date for employment at a particular company was undertaken by comparing this date with information acquired from the Workers' Compensation Mining Master card index. There was no significant difference between company reports and the MMF for employment starting dates.

Duration of employment based on company reports and on self reports in the MMF also showed no significant differences.

On the evidence provided by these validation tests, the Mining Master File accurately captures the employment experience of Ontario gold miners in the study period. The WCB are to be commended on the success of their efforts over the years to computerize and faithfully record the employment experience of workers in the Ontario mining industry.

2.2 VALIDITY OF DEATH RECORD CERTIFICATION

A critique of the 1986 Muller study questioned whether some or all of the increase in lung and stomach cancers might had been due to misdiagnosis, or at least different diagnostic practices for the gold miners than for the province as a whole (the comparison group). The cases of cancer from the Muller Study were therefore identified, and the Ontario Cancer Registry was asked whether it had additional confirming evidence of the cancer and the site of cancer for these miners as well as for all males in the Province as a whole. The Registry has recently been updated and contains very high quality information on cancers occurring within the province from 1964 onwards. It has obtained data from a variety of sources - death certificates, hospital records, pathology data, and cancer clinics.

Table 3 shows a comparison between the diagnosis on the death certificate and the final diagnosis given by the Cancer Registry based on all sources of information available. The cases sent to the Registry were those occurring among gold miners as defined in the 1986 study by Muller. Not all were found in the Registry files and the final column of Table 3 shows the numbers that were identified. Of the 120 miners with lung cancer on whom there was additional evidence, 97% were confirmed as lung cancer. This compares with 93% for all Ontario cases. In addition, there were two cases stated on the death certificates to be due to "other" types of cancer, but which were listed by the Registry to be cases of lung cancer.

For stomach cancer, just 29 of the 54 cases were identified in the Provincial Registry. This was a smaller proportion than for lung cancer. There was additional information on 28 cases, of which 24 (86%) were confirmed to be stomach cancers. This compares with 89% in the male population in Ontario. Importantly, only one of the stomach cancers was relabelled as a cancer of another site of the digestive system. Among the other digestive cancers the numbers were similar, with 86% being confirmed as at the site noted. The remainder were reallocated to cancers at "other" sites (i.e., non-digestive and non-lung). Among cancers at these other sites, 95% among gold miners were confirmed by the Provincial Registry, compared with 96% in the general population.

A slightly different presentation of the data is given in Table 4. This shows, for those cases found in the Cancer Registry files, a comparison between the total numbers of cases identified through death certificates only and those identified through the best evidence available. In general, lung, stomach and other digestive cancers were slightly over-estimated through death certificate data, but the percentage differences were very similar for both the gold miners and Ontario males. This was also true for cancers at other sites which were somewhat understated on death certificates.

SECTION 3

MORTALITY ANALYSIS OF THE REDEFINED GOLD MINING DATA

3.1 COHORT DEFINITION

Because of the concerns noted in Section 1.1, a different definition of a "gold miner" was adopted: any man who had a total of five years of dust exposure in gold mining, of which at least half a month occurred in an Ontario gold mine between 1955 and 1977, and who was x-rayed at some time in that period (dust exposures were defined according to the codes outlined in Table 5). A miner was excluded if he had had any uranium mining or milling or asbestos exposure before reaching five years gold mining or 1955 whichever was first. If asbestos or uranium exposure occurred later, person years were counted 2only up to the date when such began. In essence, this definition treats the new analysis as one of gold mining. It ignores any effects of other mining (apart from uranium). This assumption was considered reasonable in view of the absence of excess risk among other miners in the 1983 study (as well as in other studies of Ontario miners). Initial analyses checked this assumption by examining the effect on the results of omitting miners of different types. Table 6 shows the effect on the total person years of observation.

Before examining the data, certain effects of the new definition of a gold miner could be predicted. Virtually all those previously labelled as "gold miners" would be included in the new cohort. In addition, many men previously labelled as having "mixed ore exposure" would be in the cohort. Also, some surface workers in dusty occupations would be included. It was also possible that a very few miners previously labelled as, for example, "nickel/copper miners", would also be included. Overall, the number of "gold miners" would be larger than in the Muller study.

3.2 RESULTS

Table 7 shows the observed and expected deaths for the complete gold cohort by major causes of death (the table is analogous to "Table 1" in the 1986 Muller report). All the numbers of deaths are, as anticipated, larger. The SMRs change little (in particular, the stomach and lung cancers are still significantly increased).

There were only small differences in the SMRs for cohorts in which miners with other ore exposures were excluded (Table 8). For lung cancer there was a slight decline when miners with "other" exposure were excluded, but the difference was not significant. A review of the exposure for these "other" miners found that some was of "unknown" type of ore. A relatively high proportion of the unknown exposures occurred a long time ago. Some may well have been in gold mining. Thus, among those men labelled as having "other" exposure may have been some with additional gold mining experience at a time when exposures in such mines were highest. If they were at highest risk, then their exclusion will have led to a reduced SMR. However, we should recognize that, if this is so, the length of gold mining exposure in the earlier periods will be under-estimated in our data. This will slightly accentuate any dose-response relationship. Because there was little difference between the cohorts, the focus of the remainder of the analyses will be on cohort 1 - all gold miners with five years or more exposure.

Lung Cancer

We first examine lung cancer. Analysis by duration of exposure (Table 9) shows no particular pattern, and the SMR is raised even among those with relatively short (5 - 9 years') duration of exposure. However, analysis by date of first exposure (Table 10) and by time since first exposure (Table 11) show very marked trends. Those first exposed before 1936 and those exposed more than 30 years ago have more than a 60% excess of observed over expected deaths, although there is no increase in the SMR for those who first worked in a gold mine after 1950. Notably, there is also a tendency for the SMR within the early period following first exposure to be reduced, suggesting a healthy worker effect.

Since these three variables are necessarily inter-related, further analyses have been conducted examining SMRs by two of the three variables simultaneously (Tables 12, 13, and 14). With some exceptions (perhaps due partly to small numbers) the patterns are consistent with an occupational relationship. The consistencies of the relationships are perhaps as strong as can reasonably be expected in this type of data.

One concern with the use of duration of dust exposure as a measure of the amount of exposure is that the levels of environmental contaminants have varied greatly over the years. In general dust conditions have improved, so that exposures before 1936, for example, were more intense than those in recent years. For this reason, an index of weighted duration of exposure was constructed. Years of exposure up to 1935 were given a weight of 4; years between 1936 and 1944 a weight of 3; years between 1945 and 1954 a weight of 2; and years from 1955 onwards a weight of 1. Such an index will necessarily be relatively crude, but seemed reasonable in the light of what is known about konimeter counts in gold mines over this period of time.

Essentially, the index takes account of the fact that levels of contaminants were higher in the past than they have been more recently. The index also has the advantage of combining two of the three variables considered above, namely, duration of exposure and date of first exposure. The mortality patterns in different subgroups of weighted duration are shown in Table 15. A very clear trend is observed, with the SMRs increasing steadily with increasing weighted duration (Figure 2 shows the excess relative risk of lung cancer versus weighted duration of exposure). The lung cancer mortality by this index and by time since first exposure is shown in Table 16. The pattern noted above is confirmed, with a general tendency for the SMR within weighted exposure category to be greater the longer the time since first exposure, consistent with an occupational relationship.

An alternative approach to this question is shown in Table 17. This examines lung cancer mortality by duration of gold mining before 1945. It is worth noting that all miners with more than zero exposure before 1945 will have had a minimum of 10 years since first exposure, since person years were only counted from 1955 onwards. Once again, a clear trend of increasing SMR with years of exposure before 1945 was observed.

Stomach Cancer

The overall stomach cancer mortality was roughly 43% greater than expected (Table 7). This was examined further by considering mortality by duration of exposure, by calendar date of first exposure, and by time since first exposure (Tables 18, 19, 20). In general, these breakdowns show the opposite pattern to that expected. Mortality was most increased in men exposed for between 10 and 14 years, while those exposed for more than 25 years showed only a slight increase of observed deaths over expected (Table 18). Similarly, the peak SMR was noted for men exposed 10 to 19 years after first exposure while there was no increase 30 or more years after first exposure (Table 19). Further, those men first exposed more recently (1950 onwards) had the highest SMR. Indeed, the earlier the date of first exposure, the lower the SMR (Table 20). These patterns were clearly inconsistent with an expected occupational relationship, so the data were further examined by considering two variables at a time (as was done for the lung cancer data). However, these breakdowns (shown in Tables 21, 22, 23) do not explain the results further.

Additional analysis of the stomach cancer data was achieved by the same weighting of duration of exposure as was conducted for lung cancer (Table 24). The same weighting scheme was used. Once again, there was no indication of a trend, indeed the SMR tended to be lower for higher values of the index of weighted duration. Examination of the data by weighted exposure and by time since first exposure (Table 25) provided no further insights into the mortality pattern. This was also true when stomach cancer mortality by duration of gold

mining before 1945 was examined (Table 26). In general the SMR declined with duration of mining before that date, although there was a slight, non significant, increase for miners exposed for more than 20 years before 1945.

3.3 FURTHER ANALYSES

Concern was noted earlier about possible misdiagnosis of lung and/or stomach cancers. Tables 27, 28 and 29 show the differences between observed and expected numbers of these cancers as well as those of other sites of the digestive system and all remaining cancers by three variables: duration of exposure, date of first exposure and time since first exposure. There was some tendency for excesses in lung and stomach cancer to be accompanied by deficits in cancers at other sites, but there was also a pattern showing increasing excess of all cancers with increasing time since first exposure and earlier date of first exposure. Combined with the data shown in Section 3.2, this suggests that the excess of lung cancer was not due to inaccurate diagnosis.

The choice of comparison group in any occupational study is always a difficult question. Two points have been raised in relation to the gold miners - firstly that cancer rates in Northern Ontario tend to be different from those in the remainder of the province and, secondly, that there is a high proportion of immigrants among miners, and immigrants to the Province of Ontario are known to have high rates of stomach cancer (16). Further analyses were therefore conducted using Northern Ontario as a comparison group, and dividing the cohort by place of birth. Table 30 shows the effect on lung cancer mortality. (Since there was some doubt as to whether Nipissing should be included in Northern Ontario, analyses were conducted including and excluding this region but the differences were minimal so Nipissing was included). In general, the SMRs are reduced, particularly for those miners born outside Canada and the U.S. Overall, however, the number of observed deaths is still significantly greater than expected.

The same adjustments for stomach cancer (Table 31) produce even greater reductions in the SMRs. Indeed the overall SMR is no longer significant although it was 14% greater than expected (the concern as to whether this analysis may have constituted an over-adjustment will be addressed in the discussion). Surprisingly, the stomach cancer SMR was higher for Canadian and U.S. born miners than for others.

SECTION 4

DISCUSSION

The data have been examined in relation to two major concerns - the increase in lung cancer and the increase in stomach cancer found by Muller among gold miners. These will be addressed in turn.

4.1 LUNG CANCER

The detailed analysis conducted by Muller and his colleagues comprised mainly a case-control study of the lung cancer cases. This report extends the analyses of a somewhat different group of workers. The redefined cohort showed almost no difference in the SMR for lung cancer compared with the Muller Study. The various further analyses showed (within reasonable limits) the expected pattern of an occupational risk. A particularly clear dose-response relationship was noted with weighted duration of exposure, which was devised to estimate the cumulative exposure of each gold miner. These analyses therefore confirm the data of Muller and his colleagues and suggest that the increase in lung cancer is due to exposure in gold mining. One factor that has not been incorporated into the additional analysis is smoking. However, the data from Muller show that the rate of smoking among gold miners was virtually identical to that in nickel and copper miners (at least in those miners for whom smoking data could be obtained). This latter group showed no increase in risk from lung cancer. Moreover, the case-control study described in Section 1.1 found that the risk of lung cancer persisted even after adjustment was made for smoking habits. Thus, it seems that smoking has not been a confounder in these data.

The special panel was asked to consider a ranking of cases of disease according to the likelihood that they were occupationally related. The best data to achieve this for lung cancer appear to be those in Table 15. This shows lung cancer mortality by weighted duration of exposure, a measure that incorporates both the length of exposure and the intensity of exposure. Such an index makes biological sense and indeed produces a very clear and consistent dose-response relationship. It is therefore reasonable to infer that the higher the weighted duration of dust exposure of any case of lung cancer, the greater the probability that it was related to exposure in gold mining.

4.2 STOMACH CANCER

The data for stomach cancer are puzzling. There is an overall significant increase in this disease, which is apparently not accounted for by diagnostic misclassification. However, attempts to look for a dose-response relationship by duration of exposure, by calendar date of first exposure, by time since first exposure, by

combinations of these measures as well as by weighted duration of exposure are all inconsistent with an occupational relationship and indeed generally show the opposite pattern to that expected. It is clearly tempting in the light of these data to dismiss the relationship as a fluke. However, this would not explain the overall increase in disease. The explanation may be, at least in part, provided by Table 31 which shows stomach cancer mortality by place of birth and using Northern Ontario as a reference population. While some increase in the disease remains (apparently confined to those born in Canada and the U.S.) the increases are no longer statistically significant. Once again, as with lung cancer, there may be an element of over-adjustment in this computation. Nevertheless, one cannot on the basis of the evidence conclude that the increase in stomach cancer has been occupationally caused by exposure in gold mining.

4.3 OTHER COMMENTS

A number of other factors should be considered, in particular those which may have led to under- or over-estimates of the SMRs. It appears unlikely that diagnostic artifacts led to any error in the calculation of SMR for lung cancer, since the degree of misclassification appears to be similar in the gold miners and the male population of Ontario (which was used as the comparison group). Two other factors examined were the choice of comparison group and place of birth. Table 30 showed that, when mortality was compared with Northern Ontario rather than the province as a whole, the SMRs for lung cancer were reduced. One might, though, be concerned as to whether this constituted an over-adjustment - since gold miners (and possibly other occupational groups at risk) form part of the Northern Ontario population and may somewhat dilute any association. Census data show that miners formed 14% of the working population in Northern Ontario in 1961 and 10.7% in 1976. It is uncertain how many of these had worked in a gold mine, but the number is unlikely to have had a substantial effect on the comparison. Any over-adjustment will have been small. On the other hand, it is known that the computerized record linkage procedure at Statistics Canada is not perfect. Some deaths that will have occurred may be missing either because of limitations in the linkage procedure, errors in one of the files matched or because a death may have occurred outside the country. Thus Muller and his colleagues reported a comparison showing a 6% shortfall in mortality determined via record linkage among pensioners at a nickel mining company in Ontario. In addition, there may have been some healthy worker effect. Fox and Collier (17) have shown that this does indeed apply to cancer, although not as strongly as for some other diseases. Estimates of the effect vary and indeed may be highly dependent on the specific situation.

To some extent then factors that might systematically over- or under-estimate mortality, may have cancelled each other out. Insofar as this is true, the estimates given in the data above give a good indication of the increased risk of lung cancer faced by gold miners in this cohort. It should be remembered that there will have been many other gold miners who worked in the earlier, dustier conditions who left the industry before 1955 and will therefore not have been included in the cohort. It should also be noted that any estimate is subject to random error. That is, the true value may be higher or lower than that observed. The 95% confidence interval (loosely interpretable as a range of values within which we can be 95% sure that the true level of risk falls) is from 1.23 to 1.61 for lung cancer mortality. Thus the true risk from lung cancer may be as low as 23% above normal, or as high as 61% greater than expected.

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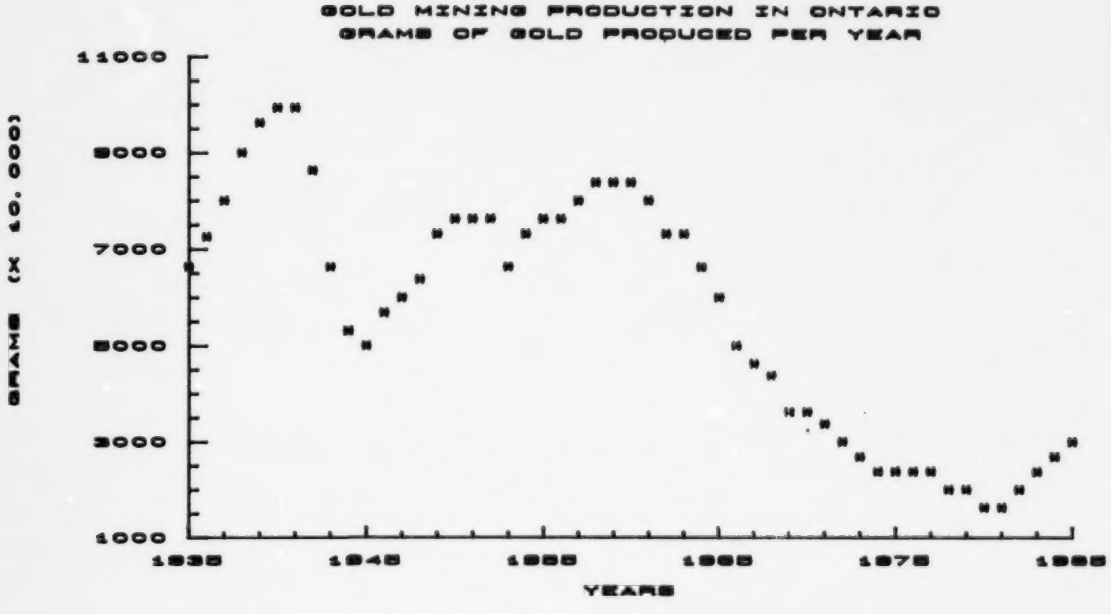
FIGURE 1

GOLD MINING PRODUCTION IN ONTARIO

Adapted from: 1985 Ontario Mineral Score. Toronto: Ontario Ministry of Northern Development and Mines, 1986 p. 194



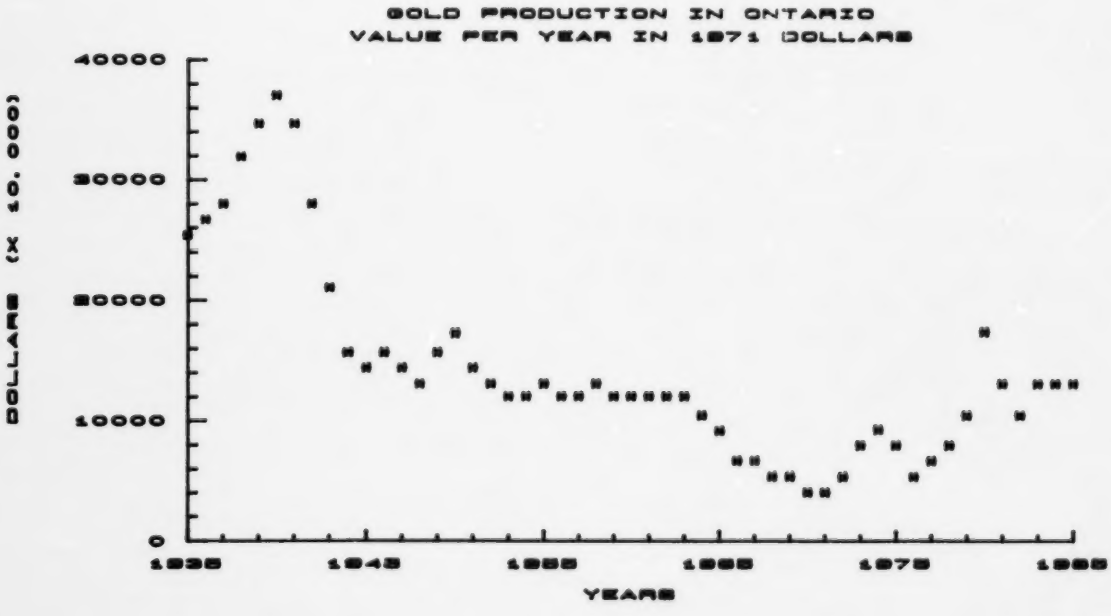


FIGURE 2

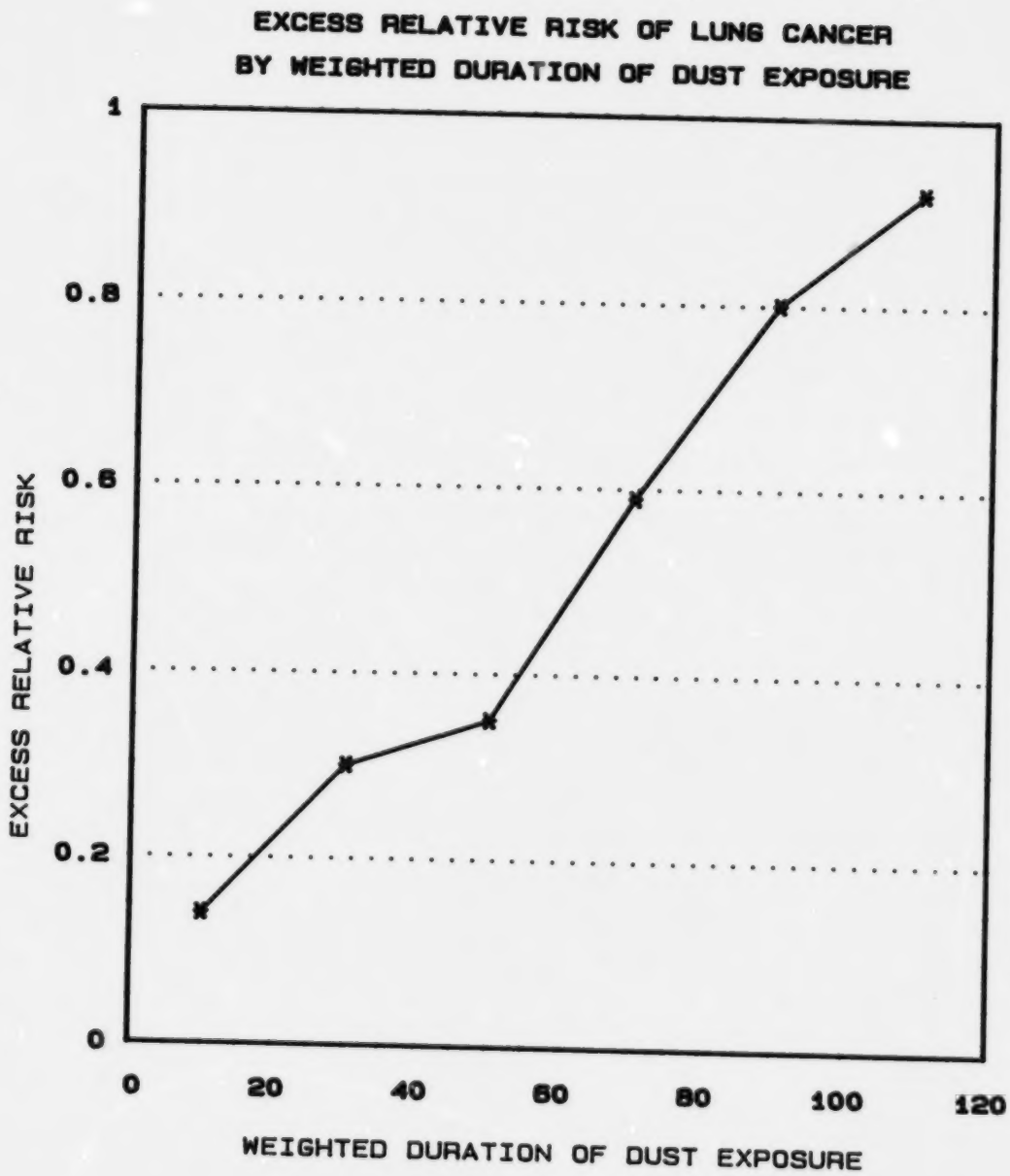

EXCESS RELATIVE RISK OF LUNG CANCER
BY WEIGHTED DURATION OF DUST EXPOSURE
EXCESS RELATIVE RISK
1
0.8
0.6
0.4
0.2
0
0
20
40
60
80
100
120
WEIGHTED DURATION OF DUST EXPOSURE

TABLE 1

LUNG CANCER MORTALITY IN GOLD MINERS

COUNTRY	Cohort size: Follow-up	a) OBS	b) EXP	c) SMR	d) 95% C.I.	e) P-Value
AUSTRALIA (10)	1974 14 years	59	40.8	140	-	0.01
SOUTH AFRICA (11)	3971 9 years	39	24.2	161	115-220	-
FRANCE (12)	1000 11 years	66	-	1034	-	-
UNITED STATES (13) Homestake, S. Dakota	3328 26 years	43	42.9	100	73-135	-
UNITED STATES (14) Lead, S. Dakota	440 14 years	10	2.7	370	-	0.01
SOVIET UNION (15)	- 27 years	-	-	f) RR=7.9	-	<0.001

a) OBS = Observed number of deaths in the cohort
b) EXP = Expected number of deaths in the cohort
c) SMR = Standardized Mortality Ratio
d) 95% C.I. = 95% Confidence Interval about the risk estimate(SMR)
e) P = Probability of obtaining the risk estimate by chance
f) RR = Relative Risk

TABLE 2

STOMACH CANCER MORTALITY IN GOLD MINERS

COUNTRY	Cohort size: Follow-up	a) OBS	b) EXP	c) SMR	d) 95% C.I.
AUSTRALIA (10)	1974 14 years	4	9.8	40	-
SOUTH AFRICA (11)	3971 9 years	11	7.0	157	78-281
FRANCE (12)	1000 11 years	e) ND			
UNITED STATES (13) Homestake, S. Dakota	3328 26 years	5	8.8	57	18-133
UNITED STATES (14) Lead, S. Dakota	440 14 years	ND			
SOVIET UNION (15)	- 27 years	-	-	f) RR=5.7	-

a) OBS = Observed number of deaths in the cohort
b) EXP = Expected number of deaths in the cohort
c) SMR = Standardized Mortality Ratio
d) 95% C.I. = 95% Confidence Interval about the risk estimate(SMR)
e) ND = Not Done
f) RR = Relative Risk

TABLE 3

AGREEMENT OF DEATH RECORD INFORMATION WITH ONTARIO CANCER REGISTRY INFORMATION

DEATH CERTIFICATE			CANCER REGISTRY: Lung Cancer		Stomach Cancer		Other Digestive Cancer		Other Cancer		SUB TOTALS	Death Certificate Only		TOTALS
	Lung Cancer	G*	116	97%	1	1%	0	-	3	2%	120	7	6%	127
		P*	19935	93%	27	-	189	1%	1372	6%	21523	1783	8%	23306
	Stomach Cancer	G	0	-	24	86%	1	4%	3	10%	28	1	3%	29
		P	0	-	5429	89%	296	5%	401	7%	6126	769	11%	6895
	Other Digestive Cancer	G	0	-	0	-	37	86%	6	14%	43	5	10%	48
		P	189	1%	304	2%	16985	89%	1713	9%	19191	2386	11%	21577
	Other Cancer	G	2	3%	0	-	1	2%	62	95%	65	5	7%	70
		P	512	2%	73	-	521	2%	28814	96%	27920	2793	9%	32713
	TOTAL	G	118	46%	25	10%	39	15%	74	29%	256	18	7%	274
		P	20636	27%	5833	8%	17991	23%	32300	42%	76760	7731	9%	84491

* NOTE: G = GOLD MINERS

P = ONTARIO MALE POPULATION

TABLE 4

DIFFERENCES IN CANCER DEATHS BY TYPE OF INFORMATION

Cause (site)		Death Certificate	Best Evidence	Difference %
Lung Cancer	G*	127	125	+ 2%
	P*	23306	22419	+ 4%
Stomach Cancer	G	29	26	+ 11%
	P	6895	6602	+ 9%
Other Digestive Cancer	G	48	44	+ 9%
	P	21577	20377	+ 6%
Other Cancers	G	70	79	- 11%
	P	32713	35093	- 7%

* Note: G = GOLD MINERS

P = ONTARIO MALE POPULATION

TABLE 5

DUST EXPOSURES DEFINED BY OCCUPATION CODES
(Based on Worker's Compensation Board Codes)

WCB CODE	DEFINITION
11	Full time in dust exposure - Mill
12	Full time in dust exposure - Other surface
13	Full time in dust exposure - Shaft sinking
14	Full time in dust exposure - Other underground
15	Full time in dust exposure - Surface and underground
16	Full time in dust exposure - Open pit
21	Part time in dust exposure - Mill (including Mill in Open Pit)
22	Part time in dust exposure - Other surface
25	Part time in dust exposure - Surface and underground
26	Part time in dust exposure - Open pit
97	Dust exposure, specifics unknown

TABLE 6

PERSON YEARS CONTRIBUTIONS OF GOLD (EVER MINED) MINING SUB-COHORTS

COHORT	TOTAL MINERS	TOTAL PYR	PYR/MINER
EVER MINED GOLD (ALL GOLD)	10185	150181.5	14.75
EVER MINED GOLD (EXCLUDE NI/CU)	9247	133818.5	14.47
EVER MINED GOLD (EXCLUDE IRON)	10029	146622.0	14.62
EVER MINED GOLD (EXCLUDE OTHER)	7402	97711.0	13.20
ONLY MINED GOLD (PURE GOLD)	6786	87863.5	12.95

TABLE 7

MORTALITY DATA ALL GOLD MINERS

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
All Causes	2183	2302.66	95	.013
All Disease Causes	1924	2139.35	90	.000
Infective Diseases	32	16.08	199	.000
Silicotuberculosis	19	-	-	
Pulmonary Tuberculosis	8	5.94	135	
Other Tuberculosis	0	1.52	-	
All Other Infective Diseases	5	7.91	63	
Malignant Neoplasms	493	490.53	101	
Digestive Organs	161	169.85	95	
Stomach	63	44.22	143	.005
Intestine or Rectum	45	71.21	63	.002
Other Digestive	53	54.42	97	
Respiratory System	216	157.94	137	.000
Nose, Nasal Cavities, etc	1	1.27	79	
Larynx	6	6.97	86	
Trachea, Bronchus, Lung	209	148.48	141	.000
Other	0	1.23	-	
Lymphatic and Hematopoietic Tissue	35	43.15	81	.215
Other	81	119.59	68	.000
Bone	0	2.08	-	
Kidney	11	11.62	95	
Bladder	8	17.85	45	.020
Brain	10	14.35	70	
Prostate	25	33.50	75	.142
Skin	2	5.65	35	.125
All Other	25	34.54	72	.105
Diseases of the Circulatory System	1002	1267.26	79	.000
Ischaemic Heart Disease	705	929.90	76	.000
Cerebrovascular Disease	158	177.84	89	.137
Other	139	159.52	87	.104
Diseases of the Respiratory System	209	145.13	144	.000
Influenza, Pneumonia, Bronchitis, and Asthma	131	121.07	108	
Silicosis, Chronic Interstitial Pneumonia	60	7.05	851	.000
Other	18	17.02	106	.000
Accidents, Poisoning, and Violence	259	163.31	159	.000

TABLE 8

MORTALITY ANALYSIS OF GOLD MINING SUB-COHORTS

	EVER MINED GOLD									
	ALL		EXCLUDE NI/CU		EXCLUDE IRON		EXCLUDE OTHER		PURE GOLD	
CAUSE OF DEATH	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
All Causes	2183	95	1874	95	2140	95	1346	95	1179	96
All Disease Causes	1924	90	1653	90	1884	90	1186	91	1034	92
Silicotuberculosis	19	-	16	-	19	-	14	-	12	-
Malignant Neoplasms	493	101	426	101	489	102	289	96	256	99
Digestive Organs	161	95	138	95	160	96	100	97	89	99
Stomach	63	142	54	143	63	145	38	141	34	146
Intestine or Rectum	45	63	37	61	45	64	28	65	26	69
Other Digestive	53	97	47	101	52	97	34	102	29	101
Respiratory System	216	137	189	140	214	138	123	128	109	130
Trachea, Bronchus, Lung	209	141	182	143	207	142	118	130	104	132
All Other Cancers	116	71	99	71	115	72	66	66	58	67
Diseases of the Circulatory System	1002	79	864	80	980	79	642	83	558	84
Diseases of the Respiratory System	209	144	178	144	204	143	116	132	98	130
Silicosis, Chronic Interstitial Pneumonia	60	851	50	834	59	850	27	631	24	652
Accidents, Poisoning, and Violence	259	159	221	153	256	160	160	153	145	155

TABLE 9

LUNG CANCER BY DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Duration of Dust Exposure (years)	Observed Deaths	Expected Deaths	SMR
5 - 9	28	21.03	133
10 - 14	30	22.53	133
15 - 19	27	21.28	127
20 - 24	31	24.74	125
25+	93	58.88	158

TABLE 10

LUNG CANCER BY CALENDAR DATE OF FIRST DUST EXPOSURE (ALL GOLD MINERS)

Date of First Dust Exposure	Observed Deaths	Expected Deaths	SMR
< 1936	128	77.7	165
1936 - 1944	51	39.7	129
1945 - 1949	16	14.9	107
> 1950	14	16.2	86

TABLE 11

LUNG CANCER BY TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Observed Deaths	Expected Deaths	SMR
5 - 9	1	1.49	67
10 - 19	11	13.75	80
20 - 29	47	38.88	121
30+	150	94.35	159

TABLE 12

LUNG CANCER BY DURATION OF DUST EXPOSURE AND TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Duration of Dust Exposure (years)								TOTAL	
	5 - 9		10 - 19		20 - 29		30+			
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	1	67	-	-	-	-	-	-	1	67
10 - 19	5	102	6	68	-	-	-	-	11	80
20 - 29	11	158	22	141	14	86	-	-	47	121
30+	11	143	29	150	52	157	58	170	150	159
TOTAL	28	133	57	130	66	133	58	170	209	141

TABLE 13

LUNG CANCER BY YEAR OF FIRST DUST EXPOSURE AND TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Year of First Dust Exposure									
	< 1936		1936-1944		1945-1949		> 1950		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	0	-	0	-	0	-	1	76	1	67
10 - 19	0	-	2	77	3	97	6	74	11	80
20 - 29	7	94	21	145	12	118	7	103	47	121
30+	121	172	28	124	1	68	0	-	150	159
TOTAL	128	165	51	129	16	107	14	86	209	141

TABLE 14

LUNG CANCER BY YEAR OF FIRST DUST EXPOSURE AND DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Duration of Dust Exposure (years)	Year of First Dust Exposure									
	< 1936		1936-1944		1945-1949		> 1950		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	6	119	9	174	6	167	7	97	28	133
10 - 19	23	169	21	142	7	93	6	77	57	130
20 - 29	43	152	19	116	3	80	1	88	66	133
30+	56	182	2	60	0	-	0	-	58	170
TOTAL	128	165	51	129	16	107	14	86	209	141

TABLE 15

LUNG CANCER MORTALITY BY WEIGHTED DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Weighted Duration* (years)	Observed Deaths	Expected Deaths	SMR
5 - 19	27	23.73	114
20 - 39	53	40.76	130
40 - 59	47	34.75	135
60 - 79	47	29.47	159
80 - 99	27	15.01	180
100 - 119	8	4.17	192
120 +	0	0.59	-

TABLE 16

LUNG CANCER MORTALITY BY WEIGHTED DURATION OF DUST EXPOSURE AND BY TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

WEIGHTED EXPOSURE* (years)	Time Since First Dust Exposure (years) 5 - 9 OBS	SMR	10 - 19 OBS	SMR	20 - 29 OBS	SMR	30 + OBS	SMR	TOTAL OBS	SMR
5 - 19	1	67	9	95	14	139	3	112	27	114
20 - 39	.		2	48	23	126	28	152	53	130
40 - 59	.		0	-	7	89	40	149	47	135
60 - 79	.		.		3	116	44	164	47	159
80 - 99	.		.		0	-	27	181	27	180
100+	.		.				8	168	8	168

* Weighting of years of exposure:

Pre - 1936, Weight = 4
1936 - 1944, Weight = 3
1945 - 1954, Weight = 2
Post - 1954, Weight = 1

NOTE: . denotes no data in cell

TABLE 17

LUNG CANCER MORTALITY BY DURATION OF DUST EXPOSURE PRIOR TO 1945 (ALL GOLD MINERS)

Pre-1945 Duration (years)	Observed Deaths	Expected Deaths	SMR
0	30	31.13	97
< 5	35	24.21	145
5 - 9	48	33.80	142
10 - 19	84	52.42	160
20 - 29	12	6.81	176
30 +	0	0.11	-
Total	209	148.48	141

TABLE 18

STOMACH CANCER MORTALITY BY DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Duration of Dust Exposure (years)	Observed Deaths	Expected Deaths	SMR
5 - 9	10	5.87	170
10 - 14	13	6.50	200
15 - 19	8	6.19	129
20 - 24	11	7.51	146
25+	21	18.14	116

TABLE 19

STOMACH CANCER MORTALITY BY CALENDAR DATE OF FIRST DUST EXPOSURE (ALL GOLD MINERS)

Date of First Dust Exposure	Observed Deaths	Expected Deaths	SMR
< 1936	27	25.6	105
1936 - 1944	14	10.8	129
1945 - 1949	9	3.8	236
> 1950	13	3.9	330

TABLE 20

STOMACH CANCER MORTALITY BY TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Observed Deaths	Expected Deaths	SMR
5 - 9	1	0.62	160
10 - 19	14	4.50	311
20 - 29	22	11.49	192
30+	26	27.62	94

TABLE 21

STOMACH CANCER MORTALITY BY DURATION OF DUST EXPOSURE AND TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Duration of Dust Exposure (yrs)									
	5 - 9		10 - 19		20 - 29		30+		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	1	161	-	-	-	-	-	-	1	160
10 - 19	5	343	9	296	-	-	-	-	14	311
20 - 29	3	168	9	206	10	188	-	-	22	192
30+	1	50	3	57	11	111	11	106	26	94
TOTAL	10	170	21	165	21	138	11	106	63	142

TABLE 22

STOMACH CANCER MORTALITY BY YEAR OF FIRST DUST EXPOSURE AND TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	Year of First Dust Exposure									
	< 1936		1936-1944		1945-1949		> 1950		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	0	-	0	-	0	-	1	195	1	160
10 - 19	0	-	3	247	3	254	8	381	14	311
20 - 29	4	120	8	175	6	267	4	302	22	192
30+	23	103	3	59	0	-	0	-	26	94
TOTAL	27	105	14	129	9	236	13	330	63	142

TABLE 23

STOMACH CANCER MORTALITY BY YEAR OF FIRST DUST EXPOSURE AND DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Duration of Dust Exposure (yrs)	Year of First Dust Exposure									
	< 1936		1936-1944		1945-1949		> 1950		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 9	1	66	0	-	4	392	5	265	10	170
10 - 19	3	68	8	180	3	149	7	381	21	165
20 - 29	13	130	5	118	2	260	1	471	21	138
30+	10	103	1	145	0	-	0	-	11	106
TOTAL	27	105	14	129	9	236	13	330	63	142

TABLE 24

STOMACH CANCER MORTALITY BY WEIGHTED DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

Weighted Duration* (years)	Observed Deaths	Expected Deaths	SMR
5 - 19	17	6.23	273
20 - 39	17	11.14	153
40 - 59	12	9.98	120
60 - 79	11	9.68	114
80 - 99	2	5.28	38
110 - 119	4	1.63	245
120 +	0	0.28	-

* Weighting of years of dust exposure: Pre - 1936, Weight =4
1936- 1944, Weight =3
1945- 1954, Weight =2
Post- 1954, Weight =1

TABLE 25

STOMACH CANCER MORTALITY BY WEIGHTED DURATION OF DUST EXPOSURE AND BY TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Weighted Duration* (years)	Time Since First Dust Exposure (years)									
	5-9		10-19		20-29		30+		TOTAL	
	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR	OBS	SMR
5 - 19	1	160	10	362	6	266	0	-	17	273
20 - 39	.		4	239	10	201	3	67	17	153
40 - 59	.		0	-	6	203	6	86	12	120
60 - 79	.		.		0	-	11	130	11	114
80 - 99	.		.		0	-	2	38	2	38
100+	.		.		.		4	210	4	210

*Weighting of years of exposure:

Pre - 1936, Weight = 4
1936 - 1944, Weight = 3
1945 - 1954, Weight = 2
Post - 1954, Weight = 1

NOTE: . denotes no data in cell

TABLE 26

STOMACH CANCER MORTALITY BY DURATION OF DUST EXPOSURE PRIOR TO 1945 (ALL GOLD MINERS)

Pre-1945 Duration (years)	Observed Deaths	Expected Deaths	SMR
0	22	7.75	284
< 5	9	6.57	137
5 - 9	10	9.60	104
10 - 19	17	17.28	98
20 - 29	5	2.93	170
30 +	0	0.09	-

TABLE 27

DIFFERENCES BETWEEN OBSERVED AND EXPECTED DEATHS (O-E) FOR VARIOUS CANCERS BY DURATION OF DUST EXPOSURE (ALL GOLD MINERS)

	SITE OF CANCER			
Duration of Dust Exposure (years)	Lung	Stomach	Other Digestive	All Other Sites
5 - 9	+ 7.0	+ 4.1	- 10.4	- 7.8
10 - 14	+ 7.5	+ 6.5	+ 1.2	- 6.2
15 - 19	+ 5.7	+ 1.8	- 8.5	- 5.5
20 - 24	+ 6.3	+ 3.5	+ 1.1	- 6.3
25+	+ 34.1	+ 2.9	- 11.0	-21.0

TABLE 28

DIFFERENCES BETWEEN OBSERVED AND EXPECTED DEATHS (O-E) FOR VARIOUS CANCERS BY TIME SINCE FIRST DUST EXPOSURE (ALL GOLD MINERS)

Time Since First Dust Exposure (years)	SITE OF CANCER			
	Lung	Stomach	Other Digestive	All Other Sites
5 - 9	- 0.5	+ 0.4	- 1.6	- 0.3
10 - 19	- 2.7	+ 9.5	- 5.1	-10.4
20 - 29	+ 8.1	+ 10.5	- 8.5	- 8.5
30+	+ 55.6	- 1.6	- 12.5	-27.5

TABLE 29

DIFFERENCES BETWEEN OBSERVED AND EXPECTED DEATHS (O-E) FOR VARIOUS CANCERS BY DATE OF FIRST DUST EXPOSURE (ALL GOLD MINERS)

Date of First Dust Exposure	SITE OF CANCER			
	Lung	Stomach	Other Digestive	All Other Sites
< 1936	+ 50.3	+ 1.4	- 15.4	-26.6
1936 - 1944	+ 11.3	+ 3.2	- 5.7	- 8.5
1945 - 1949	+ 1.1	+ 5.2	- 6.8	- 4.2
> 1950	- 2.2	+ 9.1	+ 0.3	- 7.5

TABLE 30

LUNG CANCER MORTALITY BY PLACE OF BIRTH USING TWO ONTARIO COMPARISON GROUPS (ALL GOLD MINERS)

Place of Birth	Observed Deaths	SMR USING AS COMPARISON GROUP	
		Northern Ontario*	Ontario
Canada/U.S.A.	105	132	150
Non-Canada/U.S.A.	96	118	133
Unknown	8	114	128
TOTAL	209	124	141

TABLE 31

STOMACH CANCER MORTALITY BY PLACE OF BIRTH USING TWO ONTARIO COMPARISON GROUPS (ALL GOLD MINERS)

Place of Birth	Observed Deaths	SMR USING AS COMPARISON GROUP	
		Northern Ontario*	Ontario
Canada/U.S.A.	30	121	149
Non-Canada/U.S.A.	26	92	117
Unknown	7	292	372
TOTAL	63	114	142

* Northern Ontario is defined as including the counties of: Algoma, Cochrane, Sudbury (with Regional Municipalities), Thunder Bay, Nipissing, Timiskaming, Rainy River, and Kenora.

APPENDIX B

EVIDENTIARY SOURCE INFORMATION

ON THE

ONTARIO GOLD MINING INDUSTRY

APPENDIX B

EVIDENTIARY SOURCE INFORMATION FOR THE ONTARIO GOLD MINING INDUSTRY STUDY

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APPENDIX C

REPORT OF THE MILLER ET. AL SCIENTIFIC PANEL

REPORT OF THE SCIENTIFIC PANEL ON "MORTALITY FROM CANCER AMONG ONTARIO GOLD MINERS 1955-1977"

INTRODUCTION

In December 1986 we were approached to form a special scientific panel to assess the findings from a Study on Cancer among Ontario Gold Miners 1955-1977. The initial approach was to Dr. Miller who agreed to set up a small scientific panel to assess various aspects of this study. Dr. Miller met with Dr. Kaegi, Vice President Policy Planning and Specialized Services of The Workers' Compensation Board of Ontario on December 10, 1986 and agreed on the approach to be adopted to set up the panel and for it to function efficiently. In a letter dated December 16, 1986 to Dr. Miller, Dr. Kaegi regretted the absence of formal letters of appointment but provided a copy of a letter dated August 18, 1986 addressed to Dr. James Ham, Chairman of the Industrial Disease Standards Panel in which the original request of the Board to the Panel was set out. We were given to understand that it was our task to evaluate the questions in that letter from the scientific viewpoint.

We have, therefore, interpreted our terms of reference to provide our opinions to the Board on the following points:

1. The nature of the association between experience in Ontario gold mines and excess mortality from lung cancer.

2. The criteria to be considered for the compensation of gold miners who have subsequently developed lung cancer. We have also regarded the additional questions asked of the panel as providing for us three further terms of reference.

3. What criteria should the WCB employ to compensate mixed ore miners who have subsequently developed lung cancer?

4. Does there exist sufficient evidence for the WCB to provide compensation benefits to gold or mixed ore miners who have subsequently contracted stomach cancer?

5. If the answer to the second question is yes, then what criteria should the Board employ to compensate this category of workers?

Dr. Miller requested additional clarification over the request relating to stomach cancer and in a letter dated December 18, 1986, Dr. Kaegi confirmed that the Workers' Compensation Board does need to reach some conclusions on the issue of stomach cancer in gold miners and requested the panel's opinion on this aspect as well as the primary issue of lung cancer.

These terms of reference have, therefore, dominated our considerations, though in our report we depart, for reasons of efficiency, from providing our opinion on these various issues in the exact order just presented. However, in our final Conclusion and Recommendations section we return to these terms of reference.

METHOD OF PROCEDURE

In her initial discussions with Dr. Miller, Dr. Kaegi gave him the authority to decide on the membership of the scientific panel. It was, however, agreed that the panel should preferably consist of no more than three members: Dr. Miller whose speciality is cancer epidemiology; another epidemiologist; and an expert in carcinogenesis.

After consulting with colleagues in Canada and the United States, Dr. Miller approached and received the agreement of Dr. D. Scarpelli, Chairman of the Department of Pathology, Northwestern Memorial University, Chicago and Dr. Noel Weiss, Chairman of the Department of Epidemiology, University of Washington School of Public Health and Community Medicine, Seattle to serve on the panel. The report which follows is our agreed response to the terms of reference. We are unanimous on its content and recommendations.

Two formal meetings of the Scientific Panel have been held, the first in Chicago on December 17, 1986 during which we agreed on our approach to the terms of reference and to the tasks we would attempt to complete and the second in Chicago on January 30, 1987. In addition we have had several telephone calls and exchanges of correspondence in order to finalize this report.

MATERIAL CONSIDERED

The principal document we considered was the report, "Study of Mortality of Ontario Gold Miners 1955-1977" by J. Muller, R.A. Kusiak, G. Suranyi and A.C. Ritchie, released as a public document by the Ontario Ministry of Labour, The Workers' Compensation Board of Ontario and the Atomic Energy Control Board of Canada, in July 1986. In addition we were provided with a copy of further statistical information appearing as an enclosure to a letter dated September 23, 1986 to Dr. Robert G. Elgie, Chairman of the Workers' Compensation Board from Dr. Muller. We also had available the original report on the study of mortality of Ontario miners 1955-1977, Part 1, May 1983 and we were provided at our request after our meeting on January 30 with detailed tabulations relating to a further breakdown of the data in the July 1986 document, and also some corrections to the material provided in the September 23, 1986 document.

We have also consulted other material. These include the various references which we cite in our report, some which we identified as a result of a Medline search, the remainder as a result of our own knowledge of the literature and the literature identifiable through the various

reports and publications we have consulted. In addition we were supplied with comments and criticisms of the July 1986 report submitted to The Workers' Compensation Board for our consideration by the United Steel Workers of America and the Ontario Mining Association. Finally we have also received a copy of Dr. Muller's notes on the brief presented by the Ontario Mining Association to the Industrial Disease Standards Panel.

NOTES ON TABLES

In referring to Tables from the specific reports or data made available to us on the experience in Ontario gold and mixed ore miners, we shall use the Table numbers applied in the relevant documents with a suffix. Table 1, 1986 signifies Table 1 in the July 1986 report. Table 1, Supplement refers to Table 1 in the supplementary data provided, subsequently amended.

However from these Tables and also from the special breakdown of sub-groups provided to us by Mr. Suranyi, we use the prefix P to indicate that this is a Table produced by us as part of our special panel review.

OBSERVATIONS ON MORTALITY FROM CANCER AMONG ONTARIO GOLD MINERS, 1955 - 1977

In judging whether or not employment in Ontario gold mines caused an increased risk of dying from cancer, the data that appeared in the report issued in May 1983 were not considered. In that report, there was no exclusion of the self-selected group of miners who continued to attend medical examinations after stopping work, a group probably at unusually high risk of death. Thus, it is appropriate to rely on the 1986 report, which is based on an analysis of the mortality experience only of men who had not retired prior to 1955, and the supplementary analyses provided to us.

I. Cancer of the Lung

As shown in Table 1, 1986, there were a greater number of deaths from lung cancer (165) than would have been expected in the general population (117.5). A similar increase in mortality was seen for the "mixed ore" miners, but not for the men whose jobs involved only part-time underground work. While the potentially more important confounding non-occupational exposure, cigarette smoking, was not measured precisely, it appears that these underground miners' excess risk of lung cancer could not be attributable to an unusually high prevalence of cigarette smoking - their smoking habits were quite similar to those of men who mined nickel and copper, and yet the latter group had no increased mortality from lung cancer.

We sought to determine whether all or only some categories of miners were at increased risk. There was a number of characteristics that did not appear to influence the magnitude of the excess mortality:

a) Overall duration of exposure (Table 4, 1986)

b) Time since first exposure. While data on this variable were not presented explicitly, one might predict that if the high mortality appeared only after a very long induction period, the largest mortality ratios should appear in the oldest men. This was not the case (Table 5, 1986; Table 3, Supplement).

c) Time since retired from mining. If the size of the increased risk associated with underground gold mining was to wane with increasing time following retirement, it might be predicted that there would be little excess risk observed in the very oldest men. However, there was evidence of an increased risk of lung cancer in older men (Table 5, 1986: Table 5, Supplement).

d) Type of contaminant present. The distribution of type of contaminant (silica, fibers, arsenic, sulfides) to which the miners were exposed was quite similar between cases of lung cancer and a random sample of other miners chosen as controls (Table 12, 1986), though the mean numbers of years with high dust exposure was substantially greater for cases than controls.

What did tend to predict an increased mortality rate from lung cancer was the time period during which a man was employed as an underground gold miner. Specifically, the increased risk was almost entirely confined to those who spent any time in gold mines prior to 1945 (Table P-1).

The actual duration of employment prior to 1945, however, did not seem to further influence the size of the increased risk (Table P-2). This was so even if the duration of exposure to mining prior to 1945 was short (20 months or less). It should be noted that in Tables P-1 and P-2 (and in most of our subsequent derived tables) we have chosen to present data from gold miners and "mixed ore" miners who mined gold underground combined. This is because for almost every issue examined the experience in relation to lung cancer for gold and "mixed ore" miners who mined gold underground was similar. We also suspect that given the pattern of employment in the mining industry, most "mixed ore" miners started their mining experience in a gold mine.

Among those men who worked as underground miners prior to 1945, the presence of chest x-ray abnormalities suggesting silicosis was associated with a particularly high lung cancer mortality rate, but

not for those who commenced mining in 1945 or later (Table P-3). However, there were very few person years of experience for those with silicosis who commenced mining in 1945 or later. Further, irrespective of x-ray category, there was no significant excess of lung cancer for those who commenced gold mining in 1945 or after.

The data suggest that underground mining probably acted to cause some of the men in the study first employed before 1945 to die from lung cancer. Support for this inference comes from the following:

a) The size of the increased mortality rate was considerable, and the increase is highly unlikely to be due to chance. It is also unlikely that much, if any, of this increase was related to confounding by a non-occupational factor such as cigarette smoking. Furthermore, the size of the increased mortality was highest in the groups of miners in whom it might have been predicted to be highest: Men exposed to the dustiest conditions (such as existed prior to the mid-1940's); and men with chest x-ray evidence of heavy exposure to dust (i.e., men with x-rays rated Ontario X-ray Code 4 or more).

b) While there are some studies in which underground miners and/or silicotics did not have an elevated death rate from lung cancer, most have found similar results to the present one (we discuss the positive and negative studies in more detail below). This argues that the association seen in the Ontario miners is less likely to be due to bias. More likely, the different findings among the various studies reflect variation in the environmental conditions within the mines in which the study subjects had been employed.

Nevertheless, for the great majority of miners in the study who developed lung cancer, it is more probable than not that they would have developed it even if they had not been employed as an underground gold miner. This results from the fact that for most gold miners the "baseline" risk exceeded the added risk associated with their underground mining experience. The statistic that best describes this is the attributable risk percent, defined as the excess numbers of deaths in any category, divided by the numbers that occurred in that category, multiplied by 100 [or ((Observed deaths - Expected deaths)/ Observed deaths) x 100]. For those first employed prior to 1945, the attributable risk percent was 25% for those with A x-ray rating (Ontario X-Ray Code less than 4), 50% for those with B rating (Ontario X-ray Code 4) and 59% for those with C rating (Ontario X-ray Code greater than 4). Thus, only in those gold miners with lung cancer who first worked prior to 1945 and who had evidence on x-ray of severe dust exposure (i.e. Ontario X-ray Code 4 or more) had a probability of 50% or more that their cancer was mining related.

We have endeavoured to ascertain whether there is a sub-group of those with no x-ray evidence of silicosis among whom there might be a greater excess of risk of lung cancer. Table P-4 provides one result of this process. However, as for the total group of miners first employed prior to 1945 (Table P-2) there is no evidence that prolonged employment prior to 1945 conferred any greater risk than shorter employment. We also sought evidence for a different effect at different ages (Table P-5). This suggests an effect restricted to those under the age of 65, but only among those age less than 50, is the attributable risk percent greater than 50%. However, this is based on small numbers, and the excess is not significant (Chi^2 = 2.976, 95%, confidence invervals 85-348). Table P-5 also addresses the potential influence of age among those first employed in 1945 or later. The only excess is seen in those age 50-64, but the excess could quite easily have been due to chance (Chi^2 = 2.356, 95% confidence intervals 94-174). Even if this excess were real, the attributable risk percent would be only 25%, so that a lung cancer in a man in this category would most likely have occurred in any event.

In Table P-6 we display data relating mortality from lung cancer to time since first commencing gold mining (latent-period effects). For those first employed prior to 1945, the data do not permit determining when the excess first became manifest after entering employment. However, the data suggest that after 14 years from first employment the excess risk persists at approximately the same level.

For those first employed 1945 or later, no excess occurred except possibly 15-19 years after first employment. However, this excess is not statistically significant, (Chi^2 = 1.809), and even if real, the attributable risk percent is only 30%.

II. Cancer of the Stomach

Among the gold and mixed ore miners who ever mined gold, 66 deaths from stomach cancer occurred, in contrast to 48.5 expected, SMR 136, (Table 7, Supplement 1 revised). Some of this 36% excess risk is due to an over- representation (relative to the population of Canada used as a basis for comparison) of men not born in North America in the mining industry. (On page 48 of the 1986 report, the SMRs for Canadian and non-Canadian born men both lie below the overall SMR that does not control for country of birth). This bias is probably similar in size to a bias working in the opposite direction, the "healthy worker" effect. Since cigarette smoking is at most a weak risk factor for cancer of the stomach, any differences between the miners and the comparison population in terms of smoking habits are probably unimportant here.

While the 36% increase in the rate of stomach cancer among the gold and mixed ore miners could be an indication that their occupation played a causal role in the development of some of these cancers, there are reasons to be skeptical of a causal relationship:

a) Those with the heaviest exposures to dust, i.e., those who worked in the mines prior to 1945, had only a slight and non-significant increased mortality from stomach cancer (Table P-7). Even among those with silicosis (Ontario X-ray Code 4 or more) the excess was small.

b) In contrast to the situation for lung cancer, in which both gold and mixed ore miners experienced the increase in risk, mixed ore miners had no increased mortality from stomach cancer. This was true irrespective of year of first employment (Table 7, Supplement, revised).

c) The gold miners had an exceedingly low mortality rate from colo-rectal cancer (27 observed, 55.7 expected, SMR 48, Table 1, 1986). A similarly low risk was probably present in the mixed ore miners as well (see 1983 report). Such a deficit of deaths considerably exceeds that which could result from the "healthy worker" effect. Since it probably does not represent a true health benefit associated with mining, it could be an indication of some unusual characteristics of the men who chose to be miners above and beyond that explained by their distribution of national origin, or it could reflect errors of cause of death certification. Either of these last two explanations casts doubt as to the causal nature of the increased risk for stomach cancer associated with gold mining.

d) In most other studies of death rates among gold miners, there has not been an increase in mortality from stomach cancer. While this is not incompatible with there being a true increased risk in the Ontario gold mines, neither does it offer independent support of such a relationship (see section below).

REVIEW OF RELEVANT LITERATURE

In this section we discuss a number of reports that provide information that could help in interpreting the findings in Ontario miners with gold mining experience. Where appropriate we will make comparison with the data from the Ontario miners.

Armstrong et al (1979), conducted a study of a cohort of 1,974 gold miners and 213 coal miners. These were followed for thirteen to fourteen years. Lung cancer mortality was relatively high in the gold miners (59 deaths observed, 40.8 expected, SMR 145) but weakly and inconclusively related to the extent of their underground mining experience. This is somewhat similar to the experience in Ontario gold miners. However, the authors concluded that cigarette smoking may explain the excess of lung cancer in the gold miners studied because the prevalence of smoking in the gold miners was higher than in the coal miners, or in other men in Western Australia. There was no indication of excess risk of lung cancer in those with radiologic evidence of silicosis.

Hessel et al (1986), conducted a case control study to assess the association between lung cancer and silicosis or silica dust exposure in white South African gold miners. Two controls were matched to each case by year of birth and by smoking. Controls were selected from the same group of deaths that produced the cases, 133 matched triplets were studied. There was no evidence of increased risk of lung cancer associated with radiological silicosis, though some in relation to parenchymal silicosis at autopsy. However, with the groups identified by having silicosis at either radiology or autopsy, there was an inverse association between lung cancer risk and increasing estimated levels of dust exposure.

Brown et al (1986) conducted a historical cohort study of 3,329 gold miners who worked underground at least 1 year between 1940 and 1965 in a mine in South Dakota, U.S.A. Vital status was determined as of June 1, 1977. Forty-three deaths from lung cancer were observed, for an SMR of 100 (95% CI 73-135). The SMR was 125 (61-229) for those first employed before 1930, but subsequently no group showed any indication of increase in risk. Also analyses by length of employment or estimated dust exposure and latency showed no increased risk, except for those first employed 35 - 39 years ago (SMR 162, 85-239). Complicating the interpretation of this study is the fact that the mortality rate from lung cancer in the male South Dakota population was substantially below that of the U.S. male population, the group used as a basis for comparison. Had the South Dakota rates been used instead, the overall SMR for the gold miners would have been about 160.

Katsnelson and Mokronosova (1979), however, produced somewhat different findings from the Soviet Union. In their study, primarily designed to evaluate the risk of lung cancer among those exposed to non-fibrous mineral dusts, a relative risk of 7.9 for male underground workers of a gold mine is cited. When workers with silicosis were excluded, the relative risk fell to 3.1 but was still significantly elevated. The relative risk for surface workers of a gold mine was 1.6. The authors contrast the risk of death from lung cancer with workers occupied in mining and processing talc where the relative risk was 4.5. For workers in dinas and fire clay plants, the relative risk was also elevated.

It would seem, therefore, that experience in other countries with gold mining is not totally consistent with the findings in Ontario miners. No increased risk was found in the study of South African miners, and the

excess risk in Western Australian miners was attributed largely to cigarette smoking. Cigarette smoking was not considered in the studies in South Dakota or in the Soviet Union.

Three of these studies also provide data on stomach cancer. In the study in Western Australia (Armstrong et al, 1979) there were 4 observed deaths from stomach cancer in gold miners compared to 9.8 expected (SMR 41). Thus there was a deficit of mortality from stomach cancer. Interestingly for colo-rectal cancer the observed was 9 compared to 11.0 expected (SMR 82). Similarly in the study in the USA (Brown et al, 1986) the SMR for stomach cancer was 57 (based on 5 observed deaths) while that for colo-rectal cancer was 91 (based on 16 deaths). In the study in the Soviet Union, however, the relative risk for underground workers of a gold mine was 5.7, falling only slightly to 4.4 if silicotic patients were excluded. There was no elevated risk for surface workers of a gold mine or workers of a dinas plant, but there was excess for workers occupied in mining and processing talc and those employed in fire clay plants. No data were provided, however, on risk of deaths from other gastrointestinal tumors.

The question as to whether or not silicosis in the absence of gold mining exposure increases the risk of lung cancer is controversial (Heppleston 1985). However, in addition to the study in the Soviet Union reports on elevated risk of lung cancer among silicotics have come from several countries and from Ontario. Lynge et al (1986), linked census populations characterized by job title to mortality files in Norway and Finland and incidence data in Denmark and Sweden. An excess risk of lung cancer was found for foundry workers and Swedish iron ore miners (already recognized in other studies) and a possible increase in risk in Finnish and Danish stone cutters who were believed not exposed to other carcinogens. There was some suggestion from the stone cutters, of silica increasing lung cancer risk, but this was not conclusive. There were no data on gold miners.

Forastiere et al (1986) studied 72 cases of lung cancer and 314 deceased referents from an area of central Italy where the ceramic industry had a long tradition. By linking to a file maintained by the Italian Institute for Compensation of Occupational Diseases, it was determined that 33 cases and 104 referents were ceramic workers and that 15 and 25, respectively, were silicotics. The relative risk for ceramic workers was 2.9 (95% C.I. 1.1 - 3.5) and that for ceramic workers with silicosis 3.9 (1.8 - 8.3). The latter relative risk was unchanged after controlling for cigarette smoking.

In a study of 1026 granite workers hired between 1940 and 1971 and followed to the end of 1981, 22 lung cancer deaths were observed, 17.1 being expected from the Finnish male population (SMR 129, 95% C.I. 104 - 153) (Koskela et al, 1987). No lung cancer deaths occurred in the first 10 years of follow up (4.3 were expected) and only 1 from 10 to 14.9 years (3.3 expected). For 15 or more years since entry into granite work, there were 21 lung cancer deaths with 9.5 expected (SMR 221, 189 - 254). Silicosis was not very common in this cohort, and only one lung cancer

patient had silicosis (the total with silicosis was not reported). Smoking was not explicitly considered in the analysis, though a survey found similar smoking habits to that of other Finnish industrial male workers.

Zambon et al (1987) followed to 1984 a cohort of 1313 workers compensated for silicosis during 1959 - 1963 in Italy. An SMR of 239 (95% C.I. 186-302) for lung cancer was found, with increasing risk for years since first exposure. No lung cancer deaths occurred in the first 20 years from first exposure (though only 1.3 deaths from lung cancer were expected). For 20 to 29 years from first exposure the SMR was 131 (53 - 272), for 30 to 39 years 250 (163 - 366) and for 40 or more years 300 (212 - 415). There was no indication of increasing risk with increasing duration of exposure for smokers of 10 or more cigarettes daily, but there was increasing risk with increasing duration of exposure for non and light cigarette smokers.

In a study of 2055 pottery workers (Thomas & Stewart, 1987) an SMR for lung cancer of 137 was noted for men exposed to silica but not talc (95% C.I. 109 - 164). For men exposed to silica and non-fibrous talc the SMR was 254 (95% C.I. 218 - 289).

A study of 961 silicotics from Finland (Kurppa et al., 1986), showed an SMR of 312 (99% CI 230 - 414). For the mining sub-cohort the SMR was 436 (264 -670). The type of mining was not specified. In Sweden, a study of 284 silicotics employed in mining quarrying and tunneling showed an SMR of 538, while for 428 whose occupation was in iron and steel foundries, the SMR was 385 (Westerholm et al., 1986). In Switzerland, the causes of death of 2,399 silicotics was studied (Schuler and Ruttner, 1986). SMRs could not be determined, but the mortality odds ratio (using non-pulmonary cancers as the reference) was 241. In an earlier report of the study of Zambon et al (1987) in Italy, 1,234 silicotics were followed (Zambon et al., 1986). The SMR for lung cancer was 228 (95% CI 169 - 302) but for stomach cancer only 67 (32 - 123). In miners the SMR for lung cancer was 156 (68 - 871). In this analysis the effect of smoking was also evaluated, and the excess lung cancer mortality appeared to be restricted to current or ex-smokers. A study in Austria of 1,630 workers exposed to dust including silica showed increased mortality for both lung and stomach cancer (SMRs 148 and 161, respectively) (Neuberger et al., 1986).

The studies summarized in the previous paragraph were all reported at a symposium held in April 1984 on Silica, Silicosis and Cancer. They do not comprise a complete review of the literature (which we have not attempted for this topic). Indeed it seems possible that the authors were selected for presentation at the meeting as it was known they had "positive" studies. (The Ontario study of Finkelstein et al, 1986, was also presented, but this is reviewed in more detail below). Nevertheless, there does seem to be emerging evidence to support an increased risk of lung (but not necessarily stomach) cancer in silicotics (Greenburg, 1986). Indeed some experimental evidence for the carcinogenicity of silica was presented at the same meeting (Niemeier et al., 1986; Saffiotti, 1986). The possible confounding effect of cigarette smoking was not fully addressed, though Goldsmith and Guidotti (1986) postulated a synergistic effect of combined silica exposure and cigarette smoking.

In the study of Finkelstein et al., (1986) 1,119 men receiving Workers' Compensation Awards for silicosis in Ontario since 1940, had been studied up to mid 1985. Increased mortality was found from lung cancer and stomach cancer, the SMR being 230 and 188 respectively. Adjustment for smoking and country of origin did not explain the excesses observed. It was noted that deaths from colonic and rectal cancers were fewer than expected so that overall GI cancer rates were not elevated. The workers were characterized according to the year they were first compensated for silicosis. The excess of lung cancer was noted in all those first compensated up to and including 1969, though for those first compensated 1960 - 69 the mortality excess for lung cancer did not occur until the experience in the follow-up period, 1979 - 85 was included. So far no excess in those first compensated in 1970 - 75 has been noted. For stomach cancer on the other hand the excess was largely restricted to those first compensated 1940 - 49 with mortality experience included up to the end of 1978. Thus in this group the excess for stomach cancer occurred largely in those whose first employment was almost certainly prior to 1945, no excess having occurred in those first employed in 1945 or subsequently. This is of interest because there is likely to be some overlap between the cohort studied and the Ontario gold miners cohort.

This study also included a small group of surface workers with silicosis in whom a consistent excess of lung cancer was noted and also an excess of stomach cancer in all those first compensated up to 1969.

Following a review of most of the foregoing studies, an IARC working group recently concluded that there is limited evidence for the carcinogenicity of crystalline silica to humans and sufficient evidence for the carcinogenicity of crystalline silica to experimental animals (IARC, 1987).

CONCLUSION AND RECOMMENDATIONS

1. In this section we return to our terms of reference as set out in the Introduction to our report and attempt to answer each question in turn.

   Our opinion on the association between experience in Ontario gold mines and excess mortality from lung cancer.

   The question we have been seeking to address is whether the association noted, which is undoubtedly not due to chance, could be regarded as causal. We shall address this issue in relation to the criteria generally accepted for determining causality of an association in which as in the present case, chance appears to have been ruled out as an explanation. These criteria include strength of the association, consistency, whether or not a dose response relationship can be demonstrated, whether or not negative or positive confounding have been considered and ruled out and the biological plausibility of the association.

Strength: The association is not particularly strong, even when sub-groups are examined. However, it is not out of range with other associations which have been accepted as causal under other circumstances related to occupation and lung cancer.

Sir Richard Doll, in his commentary produced for the Ontario Mining Association, noted the lack of an apparent excess of malignancies generally in this cohort, and suggested that mis-diagnosis of secondary tumors as primary lung cancer, particularly in those who had radiologic evidence of silicosis, might have been responsible for the elevated SMR. Dr. Muller has addressed this issue in his notes on the brief presented to the Industrial Disease Standards Panel by the Ontario Mining Association and we are satisfied that the data available provide strong evidence against such a bias operating.

Consistency: There is little evidence on cancer in gold miners and this, as we have reviewed elsewhere in this report, is not entirely consistent. There is possibly greater evidence for consistency in the suggestion from this study that risk of lung cancer is found in those who have radiologic evidence of silicosis.

Dose response relationships: We have sought and not observed dose response relationships except insofar as gradients of risk have been noted in terms of year of first employment, the earlier the year the greater the risk, and for radiologic evidence of silicosis, the greater the degree of evidence the greater the risk. Both observations are consistent with the inference that higher exposure to dust (which may or may not have been indirectly associated with a separate causal carcinogen) resulted in a greater degree of risk.

Confounding: The most obvious potential confounder is cigarette smoking and we have concluded that this is unlikely to be a critical confounder in this study. This conclusion is based on the analyses in the 1986 report which appears to satisfactorily address this issue. Although some minor residual confounding may be present, theoretical considerations suggest that this could not explain the elevated risk in sub-groups (Blair e. al., 1985).

Under the heading of confounding it is important to consider two issues raised by external critics of the 1986 report. The Ontario Mining Association raised the issue of the appropriateness of the general population of Ontario for the calculation of expected numbers of deaths. We believe that the extent of the differences in cancer mortality by county between Northern and Southern Ontario counties documented by them in their commentary is too small for this to be an important consideration. Similarly the issue of negative confounding by the "Healthy Worker Effect" raised by the United Steelworkers of America is relatively unimportant in relation to lung or gastric cancer, and is unlikely to have obscured (or seriously minimized) an important effect.

Biologic plausibility: Of the various substances to which gold miners are known to be exposed, gold in its native form in gold mines is unlikely to be a carcinogen. There is no evidence from non-epidemiologic studies that gold salts are carcinogenic. However, we have no evidence that other known physical or chemical carcinogens are present in the gold mines to a greater extent than in, say, nickel mines where no excess lung cancer has been observed. We speculate that increased risk may have been related to a relatively brief exposure to a strong carcinogen which was infrequent but for which the likelihood of exposure was increased under circumstances of work in the industry prior to 1945 and heavy dust exposure sufficient to induce silicosis.

Until recently, the pathologic significance of crystalline silica particles has been limited to their etiologic role in the development of pulmonary fibrosis and diffuse interstitial constrictive lung disease (silicosis). New experimental studies involving rodents have identified that protracted inhalation of silica particles leads also to epithelial hyperplasia and severe dysplasia, followed by the development of squamous cell carcinoma and more rarely, adenocarcinoma of the lung (Niemeier et al, 1986; Saffiotti, 1986). Since similar lung neoplasms were obtained in experiments in which pure silica particles were administered, as well as in those utilizing commercial mineral slag samples which contained silica particles, it seems reasonable to conclude that silica particles per se are probably carcinogenic for the lung. Although the mechanism remains to be unequivocally established, the fact that a major component of the inflammatory reaction of the lung involves monocytes and macrophages suggests a possible clue. Current research indicates that in the activated state such cells generate significant levels of superoxide ($O_2^{\cdot}$) and other oxygen free radical intermediates (Weitman et al, 1985). These free radicals are highly reactive and can damage the DNA of adjacent non-transformed fibroblasts causing them to undergo transformation to the malignant state. Thus, while we cannot precisely identify the carcinogen involved in the induction of lung cancer in the gold miners, it is possible from the experimental evidence currently in hand, that silica may have been responsible for some or all of the increased risk of lung cancer.

On the basis of these criteria taken as a whole, we conclude that the evidence is sufficiently strong to regard the association between work in Ontario gold mines and lung cancer as causal. However, there are certain restrictions on this which we address in the following section.

2. Our advice on the criteria to be considered for the compensation of gold miners who have subsequently developed lung cancer.

Our advice on this issue relates to the data derived from the 1986 study and general experience on the effect of carcinogens in inducing lung cancer.

The most important general consideration is whether or not compensation should be offered to all those with mining experience in an Ontario gold mine if they subsequently develop lung cancer. We conclude that this would be inappropriate as it is clear from the evidence from the 1986 study that a substantial proportion of lung cancers would have occurred in the absence of occupational exposure to gold mining. Where relevant we have made estimates of the proportion attributable to gold mining and in only a few sub-groups does this proportion exceed 50%.

Under circumstances where proportional compensation is not offered it would generally be expected that compensation should be made available to those for whom the probability that disease would have occurred because of gold mining is greater than an even chance. This would require the attributable risk percent to be more than 50%. We have sought to identify the sub-groups in the 1986 study who comply with this criterion, and have identified only men who were first employed prior to 1945 and who subsequently had a chest x-ray rating of Ontario chest X-ray Code 4 or more.

An additional requirement would normally be applied under circumstances of Workers' Compensation, consistent with biological knowledge on carcinogenicity. This is that the time of first exposure should be a minimum number of years prior to the diagnosis of lung cancer. The data available from the 1986 study which provide guidance on this point are to be found in Table P-6, and these are sparse. However, from other studies we conclude that the normally anticipated "latent period" for induction of lung cancer is fifteen years or more. Nevertheless in accordance with the request that we should wherever appropriate "give the benefit of doubt" we would accept a minimum period of 10 years.

We, therefore, **recommend** that Ontario gold miners first employed prior to 1945 who developed lung cancer a minimum of 10 years following entry to a gold mine **and** whose x-rays were rated by the Ontario X-ray Code 4 or more be granted compensation.

We have endeavoured to identify other sub-groups among those excluded from these categories (those with an Ontario X-ray Code of less than 4, first employed before 1945 and those first employed 1945 and after) whose degree of risk might be such as to justify consideration for compensation. We have considered age at diagnosis of lung cancer, period of exposure to gold mining prior to 1945 and time since first employment to death. We have been unable to demonstrate any sub-groups with sufficiently increased risk to justify compensation.

However, we **recommend** that the question of compensation in those categories currently excluded be reconsidered from time to time especially with regard to those first employed in 1945 or after, as it is possible that continuing follow-up coinciding with continual increase in time from first employment would eventually demonstrate a significantly increased risk of lung cancer.

3. We have considered the criteria that the WCB should employ to compensate mixed ore miners who have subsequently developed lung cancer. We **recommend** that identical citeria be applied to mixed ore miners with lung cancer who had a definite history of full-time underground mining **prior to 1945** and whose x-rays were rated by the Ontario X-ray Code 4 or more, as for gold miners.

4. We were asked whether there exists sufficient evidence for the WCB to provide compensation benefits to gold or mixed ore miners who have subsequently contracted stomach cancer.

   We have concluded that sufficient evidence does not exist to provide compensation. We have commented on the unusual features of the sub-groups at increased risk and postulated that a least some of the apparent excess could be due to mis-diagnosis of cancer of other gastrointestinal sites. We, therefore, **recommend** that a special study be undertaken of the evidence for the death certificate diagonsis for those gold or mixed ore miners who have been certified as dying of a gastrointestinal cancer. However, even if a diagnostic artifact were to be ruled out we would still have difficulty in making an inference of cause and effect based upon the observed association. We note no excess risk of stomach cancer in mixed ore miners and that the observed excess occurs in a group (1945 or later employees) contrary to expectation and to experience in other circumstances (i.e. those compensated in Ontario for silicosis). We believe that the association is probably non-causal, but would be prepared to readdress the issue if further evidence is forthcoming.

ACKNOWLEDGEMENTS

We should like to express our appreciation to Dr. Elizabeth Kaegi and Dr. Neva Hilliard of the Workers' Compensation Board for facilitating our access to the material and study reports necessary for our review. We should also like to thank Mr. George Suranyi for running special tabulations to our specifications of the findings from the study of mortality of gold and mixed ore miners in Ontario.

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Chairman

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Table P-1

Lung Cancer Mortality in Relation to Year of First Employment; Gold and Mixed Ore Miners*

Year	Observed	Expected	SMR
Prior to 1935-36	127	77.4	164
1935-36 to 1944	65	45.7	142
After 1944	45	48.4	110

* Data from Table 5, 1986 and Table 2, Supp. (revised)

Table P-2

Lung Cancer Mortality in Relation to Period of Employment Prior to 1945; Gold and Mixed Ore Miners Combined*

Period (Months or Years)	Observed	Expected	SMR	Excess Deaths/1000**
0.1 - 20 months	13	8.253	157	0.52
20.1 - 40 months	19	10.026	189	1.01
40.1 - 60 months	17	11.705	145	0.59
5.1 - 10 years	55	36.983	148	0.78
10.1 - 15 years	46	31.467	146	0.93
15.1 - 20 years	31	18.420	168	1.53
20.1 + years	11	6.259	176	1.79

* Data from special tabulation provided by G. Suryani, February 3, 1987.

** Defined as: $\frac{\text{Observed - Expected Deaths}}{\text{Person-years of observation in thousands}}$

Note: The difference between the total in this table (192) and that in Table P-1 is accounted for by the exclusion of one mixed ore miner who was not exposed to underground gold mining prior to 1945.

Table P-3

Lung Cancer Mortality in Relation to
Year of First Employment and X-ray Rating;
Gold and Mixed Ore Miners With Underground Mining Experience Combined*

Year of First Employment	X-Ray Rating	Observed	Expected	SMR	95% Confidence Intervals
Before 1945	A	118	88.052	134	113 - 155
Before 1945	B	53	26.358	201	162 - 240
Before 1945	C	21	8.694	242	173 - 309
Before 1945	Total	192	123.104	156	138 - 174
1945 and after	A	44	39.521	111	80 - 143
1945 and after	B	1	2.368	42	-88 - 172
1945 and after	C	0	0.415	-	-
1945 and after	Total	45	42.304	106	76 - 137

* Data from special tabulations provided by C. Suryani, February 3, 1987.

Note: The difference between the total in this Table and that in Table P-1 is accounted for by the exclusion of mixed ore miners who never mined gold underground.

Table P-4

Lung Cancer Mortality in Relation to
Period of Employment Prior to 1945 of Those with A Rating on Chest X-Ray;
Gold and Mixed Ore Miners Combined*

Period (Months or Years)	Observed	Expected	SMR	Excess Deaths/1000
0.1 - 20 months	10	7.231	138	0.33
20.1 - 40 months	12	8.579	140	0.42
40.1 - 60 months	14	9.327	150	0.61
5.1 - 10 years	38	29.363	129	0.44
10.1 - 15 years	29	21.185	137	0.71
15.1 - 20 years	11	9.873	111	0.24
20.1 + years	4	2.494	160	1.39

Table P-5

Lung Cancer Mortality in Relation to
Age at Death for Those With A Rating on Chest X-Ray;
Gold and Mixed Ore Miners With Underground Gold Mining Experience Combined*

Age	Observed	Expected	SMR	Attributable Risk %
A: First Employed Prior to 1945				
Less than 50	5	2.31	216	54%
50-64	65	40.00	162	38%
65 or more	48	45.74	105	5%
B: First Employed 1945 or Later				
Less than 50	7	9.07	77	0%
50-64	33	24.60	134	25%
65 or more	4	5.85	68	0%

* Data from special tabulations provided by G. Suryani, February 3, 1987.

Table P-6

Lung Cancer Mortality in Relation to
Time Since First Commenced Gold Mining;
Gold and Mixed Ore Miners With Underground Mining Experience Combined*

Time Since First Mined Gold (Years)	Observed	Expected	SMR	95% Confidence Intervals
A: First Employed Prior to 1945				
10-14	3	2.423	124	-5-252
15-19	12	7.237	166	92-240
20-24	20	15.802	127	76-177
25-29	48	26.940	178	140-217
30-34	42	29.531	42	105-179
35-39	40	23.741	68	127-210
40-44	21	13.134	160	105-215
45 or more	6	3.983	151	50-251
B: First Employed 1945 or Later				
Less than 5	3	3.199	94	-18-206
5-9	5	5.895	85	2-167
10-14	11	10.225	108	45-170
15-19	17	11.867	143	85-201
20-24	8	9.413	85	20-150
25-29	1	2.157	46	-90-182

* Data from special tabulations provided by G. Suryani, February 10, 1987.

Table P-7

Stomach Cancer Mortality in Relation to
Year of First Employment and X-ray Rating;
Gold and Mixed Ore Miners With Underground Mining Experience Combined*

Year of First Employment	X-Ray Rating	Observed	Expected	SMR	95% Confidence Intervals
Before 1945	A	28	26.607	105	66 - 144
Before 1945	B	12	8.510	141	72 - 210
Before 1945	C	3	2.909	103	-14 - 220
Before 1945	Total	43	38.026	113	81 - 145
1945 and after	A	22	9.714	226	162 - 290
1945 and after	B	1	.571	175	-89 - 440
1945 and after	C	0	.130	-	-
1945 and after	Total	23	10.415	221	159 - 283

*Data from special tabulations provided by G. Suryani, February 3, 1987.

Table P-8

Stomach Cancer Mortality in Relation to
Age at Death
Gold and Mixed Ore Miners With Underground Gold Mining Experience Combined*

Age	Observed	Expected	SMR	Excess Risk/1000
A: First Employed Prior to 1945				
Less than 50	2	1.084	184	.06
50-64	21	15.154	139	.14
65 or more	20	21.787	92	-
B: 2 First Employed 1945 or Later				
Less than 50	9	3.015	298	.07
50-64	9	5.653	159	.14
65 or more	5	1.747	286	1.62

Table P-9

Stomach Cancer Mortality in Relation to
Work as Gold or Mixed Ore Miners With Underground Gold Mining Experience
By Year of First Employment*

Year of First Employment	Type of Rating	Observed	Expected	SMR	95% Confidence Intervals
Before 1945	Gold	35	28.547	123	85 - 160
Before 1945	Mixed	8	9.478	84	19 - 149
1945 and after	Gold	19	5.897	322	240 - 405
1945 and after	Mixed	4	4.519	88	-6 - 183

* Data from special tabulations provided by G. Suryani, February 3, 1987

Ontario Ministry of Labour
Ministère du Travail de l'Ontario

Industrial Disease Standards Panel
Comité des normes en matière de maladies professionelles

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April 22, 1987

MEMORANDUM TO: The Workers' Compensation Board

FROM: Linda Jolley and
Jean Louis Gagnon
Members of the
Industrial Disease Standards Panel

RE: **DISSENT FROM MAJORITY REPORT ON ONTARIO GOLD MINING INDUSTRY**

1.0 ISSUES AND FINDINGS

Consensus was reached at this time on all items in Section 1.0, with the understanding that the research on stomach cancer will be updated at the earliest possible time.

2.0 THE BURDEN OF EXCESS LUNG CANCER DISEASE

We, as panel members, cannot agree with the majority view put forward on the total burden of lung cancer experienced by gold miners in Ontario. To limit the total burden of compensable lung cancer to only those miners, whose exposures fall within the statistical excess identified through epidemiological methods, means that we ignore the very real contribution that gold mining as an occupation made to all of the lung cancer experienced by that group. After all, the case control study found that smoking was not a variable in this group's lung cancer. It is clear from the Special Panel's analyses that there is a dose- response relationship between the weighted exposures and excess risk, but there is no identifiable threshold in this data. Indeed, the dose-response curve is dramatically presented in Figure 2., showing almost a straight line through zero, which establishes that every exposure above zero contributes to these miners' risk of lung cancer. And therefore, by limiting the burden to only those identified as excess, we are ignoring this very real contribution.

As well, by limiting the burden of compensable lung cancer to only those miners, who fall within the statistical excess, we ignore legitimate claims from miners whose lung cancer contributed to the overall cancer burden in the Ontario population.

Entry into the cohort, while broadened in the Special Panel's analysis, is still limited to those miners with a minimum of 60 months in gold mining exposure who were alive at some point between 1955 and 1977 and had been x-rayed at the miners' clinic. By restricting the cohort in this way, we may have missed disease in miners with short-term exposures or those who have died before 1955 or were unable to be x-rayed. And this too can underestimate the actual burden of lung cancer experienced in this group.

The cut-off date for follow-up in all these studies was 1977, and it is clear in the analysis of disease among early miners that latency periods, from the time of first exposure to actual death, were as long as thirty years before the true extent of the cancer excess was demonstrated statistically. Therefore, to draw any conclusions about miners starting after 1955, especially to suggest that there is little or no risk, as the Eligibility Rule implies, is not only inappropriate but unfounded. Again, the clear dose-response relationship indicates no threshold and shows that every exposure in gold mining carries some risk. We have simply not had sufficient time of follow-up to demonstrate the disease, statistically.

Nor, as we stated in Section 1.0, have we dealt with the Healthy Worker effect which tends to minimize risk especially in workers at early follow-up.

At this time, we do not know exactly what causes the lung cancer nor are we prepared to limit the causative agent to silica only. Radon, arsenic and other carcinogens have been identified in these mines. Until we have a clear understanding of the causative agent or agents and their carcinogenic action, we cannot draw conclusions about which miners are even exposed to what agents, and the extent of the exposure necessary to initiate or promote cancer.

While we believe that epidemiology can identify a probable connection between lung cancer and working in gold mining, we do not believe that epidemiology is able to identify those individual workers who are suffering from an occupational disease. It is an ecological fallacy to presume that statistical studies of groups with their methodological shortcomings can produce very rigid Eligibility Rules for determining individual cases.

Therefore, at this time, we as panel members do not believe that we are able to accurately estimate the true burden of lung cancer experienced by the gold miners in Ontario.

3.0 ELIGIBILITY RULES

The Eligibility Rule proposed in the Majority Report requires an exposure weighted at 60 for any miner to be automatically eligible for compensation. And what this means in reality is neither supportable nor appropriate. The Special Panel developed a weighting system as follows:

Exposures before 1936 are weighted at 4;
Exposures between 1936 and 1944 are weighted at 3;
Exposures between 1945 and 1954 are weighted at 2;
Exposures after 1954 are weighted at 1.

Therefore to actually qualify, a miner would have to have one of the following:

15 years of exposure before 1936;
20 years of exposure between 1936 and 1944;
30 years of exposure between 1945 and 1954;
60 years of exposure after 1955;
or a combination of years at whatever exposure that will add up to 60 or more.

Indeed, a miner starting work after 1955, will never be eligible under this rule. No miner could fulfill the 60 years of exposure given our present age limits on when we can start work and when we must retire.

Once again we refer back to the dose-response relationship in the Special Panel's Figure 2 which demonstrates risk at every exposure above zero, which indicates to us that restrictve Eligibility Rules that require 15 to 60 years of exposure are not supportable. Nor, as we stated in our section on short follow-up, can a criteria of 60 years of exposure be justified in miners exposed since 1955. Indeed, both the Special Panel and Miller et al. (Table 17 and Table P-2) indicate elevated SMR's for much shorter exposures for those miners exposed before 1945. The Special Panel shows elevated SMRs for exposures of less than five years and the Miller et al. data show elevated SMRs and excess death at exposures of less than 2 years (0.1 to 20 months) and less than 4 years (20.1 to 40 months). Therefore, we do not accept that the data supports this restrictive Eligibility Rule.

Nor can we as panel members support a criteria that can never be met by any miner presently employed in our gold mines.

While the Majority Report attempts to address careful case assessment for all gold miners and especially those who do not meet the Eligibility Rule, the burden is still placed on proving the miner's claim, and therefore, ultimately on that miner or his or her family to prove their case. Miners must not only bear the burden of disease, but then they must bear the burden of proving its work-relatedness.

Since we believe that a probable connection between lung cancer and gold mining has been demonstrated, it is appropriate for the Workers' Compensation Board to use Section 122(9) of the Workers' Compensation Act for the purposes of compensating these miners. Section 122(9) states:

> 122(9) If the worker at or before the date of disablement was employed in the process mentioned in the second column of Schedule 3 and the disease contracted is the disease in the first column of the Schedule set out opposite to the description of the process, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved.

Therefore, we as panel members recommend:

ELIGIBILITY RULE: THAT LUNG CANCER AND GOLD MINING BE ENTERED INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT, AND THAT LUNG CANCER SHALL BE DEEMED TO HAVE BEEN DUE TO THE NATURE OF GOLD MINING UNLESS THE CONTRARY IS PROVED.

We would then concur with RECOMMENDATION 1. in the Majority Report that the Workers' Compensation Board meticulously search out all historical cases of lung cancer, but because the presumption would be given to the miners in these cases, the rest of the Majority Recommendations are unnecessary.